

THE POLIOMYELITIS OUTBREAK  
OF 1947 AND SUBSEQUENT YEARS  
WITH A SURVEY OF ITS EFFECTS IN  
THE COMBINED COUNTY OF MORAY AND NAIRN

by

I.C. MONRO.

Thesis presented for the Degree of M.D.



INTRODUCTION.

The year 1947 saw Great Britain involved in an outbreak of Acute Anterior Poliomyelitis more extensive than any previously experienced. In England and Wales there were 7,800 cases of poliomyelitis and polio-encephalitis notified. This gives a rate of 18 cases per 100,000 of the population, whereas the previous greatest incidence was 4 cases per 100,000 in 1938. In Scotland, the outbreak was of similar severity. Notifications numbered 1,698, of which 1,434 were confirmed, equal to a rate of 28 per 100,000 compared with the previous highest of 5.6 per 100,000 in 1928.

Some localities were heavily involved, while their neighbours were hardly affected. The Combined County of Moray and Nairn was one of the former, having, in 1947, over 50 cases in a population numbering between 50,000 and 60,000, approximating to a case incidence of 100 per 100,000. Its neighbours, Banffshire and Inverness-shire, with essentially similar populations, were relatively unaffected, the former having 2 cases, and the latter 15, while the large Burgh of Inverness, with rather less than half the population, had no cases at all. The study now presented finds its justification in the extraordinary local intensity of the outbreak.



### PLAN OF STUDY.

The study commences with a general description of the disease. This description follows orthodox lines, and is influenced by the writings of many others, in general text-books and in papers dealing with particular aspects of the subject. The main headings of the description are listed below.

Historical Survey  
Aetiology, and Epidemiology  
Pathology  
Clinical Features  
Diagnosis and Differential Diagnosis  
Treatment  
Prognosis  
Prophylaxis

After the preliminary survey of the present state of our knowledge of the disease, the outbreak in Moray and Nairn will be described under the following headings.

Occurrence of Poliomyelitis in the World  
1939-1946.  
History of Poliomyelitis in Great Britain.  
Relationship to Meteorological Conditions.  
Meteorological Conditions 1946-1947.  
Outbreak in 1947.  
Onset - location.  
- weather in locality of onset.  
Rise - spread to the various parts of the country.  
Decline.  
Scotland in Relation to England and Wales.  
Moray and Nairn in Relation to Scotland.  
1. Previous experience.  
2. Weather.  
3. Time relationship.  
4. Clinical material.  
(a) Number and nature of cases.  
(b) Symptomatology.  
(c) Comparison with other series.  
(d) Diagnosis and Differential Diagnosis.  
(e) Treatment and its results.  
5. Epidemiology.  
(a) General Picture.  
(b) Cases in the Burgh of Rothes and environs  
(c) Cases in the Burgh of Elgin and environs  
(d) Cases in the Burgh of Forres and environs  
(e) Cases in the Burgh of Lossiemouth and environs  
(f) Isolated Cases.  
(g)/

- (g) Areas not affected.
- (h) Cases in 1948.
  - i. Location.
  - ii. Nature, with special reference to vertigo.
  - iii. Relation to weather.
- (j) Lines of infection leading to Moray and Nairn.
- (k) Multiple cases.
- (l) Infectivity of cases.
- (m) Fatigue and travel in relation to morbidity.

## 6. Preventive Measures.

### Discussion.

### Sources of Information.

In addition to the standard current text-books on medicine, infectious diseases, and neurology, the following works have been freely consulted.

- "Virus Diseases of Man"  
van Rooyen C.E. and Rhodes A.J. 1948.
- "Acute Anterior Poliomyelitis"  
Rhodes A.J.  
Bulletin of Hygiene Vol: 22. No: 6. 1947.
- "A Bibliography of Infantile Paralysis 1789 - 1944"  
Fishbein, Hektoen, and Salmonsens. 1946.
- "Poliomyelitis"  
International Committee. 1932.

Without these sources of information, this study would not have been possible. As a result of their perusal, the risk of plagiarism is serious. Written sources of information must now number well over 10,000, and where a statement has once been correctly made, and clearly recorded in writing, it is difficult for those coming after not to copy the form of words used by the pioneer.

My experiences in the clinical and epidemiological field clearly indicated the need for careful study of the literature. In the first instance the search was for help in combatting a serious local situation. Gradually the inquiry spread to all aspects of the disease, including the historical. The results of the inquiry are embodied in the following pages. Clearly, no claim to original discovery can be made. I do claim, however, that, in the true literary sense, it is a research, an examination anew, with the object of appraising the value of the contributions of the many authorities, and their assembly into a coherent whole in the light of experience and judgment.

A glance at the "Bibliography" will serve to show what is entailed in making a survey of the literature concerning poliomyelitis, and to emphasise that this study cannot pretend to be such a survey. References to the literature are made where it is deemed necessary to cite an authority for the view expressed, or to pay due deference to precedence in recording observations.

## HISTORICAL SURVEY.

Historically, acute anterior poliomyelitis may be said to have passed through three main stages. In the first, the existence of the disease is to be deduced from the results of archaeological investigations. In the second, the disease receives the notice of ancient or mediaeval writers or artists. In the third, it emerges from the mists of antiquity, and receives attention from the modern medical profession, with the steady elucidation of its problems.

### Archaeology.

If the archaeologists, and the physicians they have consulted concerning their finds, are to be trusted, poliomyelitis is a disease of great antiquity. Its existence has been deduced from the shortening of a femur in a skeleton found by Flinders Petrie in Egypt, and ascribed by him to about 3700 B.C.(1). A stele, which has been freely photographed, and which appears to date from the XVIIIth Dynasty of Ancient Egypt, bears a representation of a man with a withered leg(2). There can be little doubt that the condition depicted by the artist could have resulted from poliomyelitis. A graveyard in Greenland(3), recently investigated by archaeologists, and ascribed by them to the early fifteenth century, contained skeletons with defects considered to be due to poliomyelitis, though tuberculous disease and rachitis are also mentioned. There is a long gap between Ancient Egyptian mummies and engravings, and mediaeval Norse settlements, but there are apparently no indications from archaeological sources that the disease occurred, or was prevalent in the interim. As the evidence must largely come from burying grounds this is perhaps to be expected. The preservation of the dead by climate, as in Greenland, or by artifice, as in Ancient Egypt, is not to be universally expected, or at least the remains are likely to be so disintegrated that confident deductions concerning disease of the soft parts cannot be made.

### Ancient and Mediaeval Writings and Art.

Hippocrates of Cos, born circa 460 B.C., records an outbreak of paralysis in the island of Thasos(4). This occurred in winter, after a dry summer and wet autumn. The climatic accompaniments of this outbreak vary from the characteristic late summer prevalence, and association with a continental type of climate, which have been fairly typical of the major outbreaks of this century. This appears to be the only writing concerning an outbreak of disease remotely resembling poliomyelitis. At all dates, however, there are references to the "halt, the lame and the blind",  
two/



two of these three conditions could result from poliomyelitis.

In the Gospels and in the Acts of the Apostles, there are several references to the miraculous cure of persons "sick of the palsy". In one instance, namely Acts IX. 33, there is perhaps evidence of the onset of the paralysis in childhood. The passage is rendered in the Authorised Version as follows:- "And there he found a certain man named Aeneas, which had kept his bed eight years, and was sick of the palsy." The preposition used in the Greek is ἐκ, which, with the genitive case may indicate the commencement of a period of time. That is to say, the passage may mean that Aeneas had kept his bed on account of palsy from the age of eight years. Be that as it may, there is no doubt that the paralytic was an all too frequent spectacle in the lands of the Eastern Mediterranean basin nineteen hundred years ago, and it seems not unreasonable to attribute a proportion of this maiming to poliomyelitis. In general, the artists of this, and later ages, in fact right up to the Seventeenth Century, support this view, by depicting all manner of morbid conditions, some of which have been deemed by experts who have studied them to have been portrayals of persons paralysed by poliomyelitis.

#### Poliomyelitis in Modern Times.

While this is undoubtedly the medical era, the first quotation is from a non-medical writer, Sir Walter Scott. In his autobiography, incorporated in Lockhart's "Life", he gives the following description of his illness.

"I was born, as I believe, on the 15th August, 1771.....

"I shewed every sign of health and strength until I was about eighteen months old. One night, I have often been told, I shewed great reluctance to be caught and put to bed; and after being chased about the room, was apprehended and consigned to my dormitory with some difficulty. In the morning, I was discovered to be affected with the fever which often accompanies the cutting of large teeth. It held me three days. On the fourth, when they went to bathe me as usual, they discovered that I had lost the power of my right leg. My grandfather, an excellent anatomist as well as physician, the late worthy Alexander Wood, and many others of the most respectable faculty, were consulted. There appeared to be no dislocation or sprain; blisters and other topical remedies were applied in vain. When the efforts of regular physicians had been exhausted, without the slightest success, my anxious parents, during the course of many years, eagerly grasped at every prospect of cure which was held out by the promise of empirics/

empirics, or of ancient ladies or gentlemen, who conceived themselves entitled to recommend various remedies some of which were of a nature sufficiently singular."

In view of Scott's descent from a leading Edinburgh physician, a pupil of Boerhaave, and access to the profession as a whole, the passage quoted may be accepted as a synopsis of the medical knowledge of the city at the end of the eighteenth century, and the beginning of the nineteenth. The disease was clearly a curiosity, the nature of which was not understood. The late Professor Gulland used, in the third decade of this century, to quote this passage to his students as an exact description of poliomyelitis, and indeed it can bear no other interpretation. Who first recognised the disease from this description is not clear, but the idea appears to have gained wide currency. Both John Buchan and Una Pope-Hennessy, in their works on Scott, published in 1932, state without reservation that the condition was infantile paralysis, which term had by then taken on the explicit meaning of poliomyelitis. True, writing in the "Nation" in 1916, makes the same assertion, and the matter is also touched on by Garrison, in the Journal of the American Medical Association, 1919.

One minor point does not seem to have been commented on previously, namely that if Scott were accurate in stating his age to be eighteen months at the time of the attack, this took place in February, 1773. Present day experience of the disease is that a winter occurrence is unlikely. Grierson, however, in the Editor's Preface to the Letters of Sir Walter Scott writes, "In the matter of dating letters we have done our best, but must ask for some leniency of criticism. Scott's only dates are often "Tuesday", "Friday", "11th June", etc; and, if he gives a more precise date, it is not infrequently a wrong one. He has no scruples about giving a thirty-first day to June or September and a September letter in March." If he were so inaccurate about dates which he could have known accurately, what more likely than substantial inaccuracy in dating an event of which he had only second-hand knowledge. The account clearly describes a toddler. The illness was associated with teething, and it seems reasonable to suggest that the age of eighteen months might be associated in the Author's mind with the cutting of teeth, which commonly occurs about that age. I suggest, therefore, that his illness most likely occurred later in 1773, say late June or early July, and that such a date is not inconsistent with the known facts of Scott's case, nor with present-day epidemiological experience.

From Scott to Badham, the temporary omission of some other writers being justified by the great similarity/

similarity between the two descriptions. To Badham undoubtedly goes the credit of making the first observations on a series of cases recorded in modern times. His four cases were all young children. Three suffered from paralysis of the lower limb, and one of the upper limb. One of the patients also had a transient ocular palsy. Badham's paper was published in the London Medical Gazette for 1834-35, so that the cases must have occurred not later than 1834, and possibly 1833. He saw his first patient on August 14th, the onset having been about two days previously, and the remainder apparently, though he does not say so, within the next few weeks. It seems quite reasonable to assume that all four cases occurred in a circumscribed locality. There is no doubt that the time of year is that usually found nowadays.

In the years between Scott and Badham, several reports(5) of paralytic disease appeared. The emphasis seems to have been on the spinal curvature induced by paralysis rather than on the nature of the paralysis itself, though in at least one instance the association with disease of the anterior part of the spinal cord is noted. An interesting side-light is the suggestion that the disease is more commonly met in young Anglo-Indian children, and that it may have been carried to the west as a result of the traffic between Europe and India in the eighteenth century.

Heine(6) described the disease in 1840, and stressed the following as its characteristics - selective incidence in young children; lack of apparent predisposing causes, a preparalytic stage, pain in paralytic areas, and the relative absence of cerebral symptoms. He located the site of the lesion in the anterior part of the spinal cord, and propounded a hypothetical pathology. He does not appear to have considered the disease to be communicable.

Medin(7) was the first to record an outbreak on a substantial scale. He recorded forty-four cases which occurred in the summer of 1887. Rather over a third of these cases were atypical in that they did not fit in with Heine's description. Medin, however, noted that they shared the same symptoms of onset, and that they occurred simultaneously with the typical cases. He therefore considered that he was dealing with a clinical entity, poliomyelitis anterior acuta, the nature of the symptoms depending on the part of the central nervous system affected. In his view, the condition was to be numbered amongst the communicable diseases.

Shortly after the turn of the century, the serious outbreak of 1905 occurred in Sweden. Its chronicler, Wickman/



Wickman(8), noted the occurrence of abortive, non-paralytic, and paralytic types of the disease, and extended their descriptions. In the sparsely populated Swedish countryside, where contacts with the outside world are few, and well-remembered by those concerned, he was able to show that case to case spread by contact was usual, that contact through an apparently healthy person was sufficient to account for the spread of infection, and that much spread could be attributed to abortive and missed cases.

Before leaving the period of the earlier moderns, I venture to indulge in a little family history. My grandfather and his sister were simultaneously attacked by an illness which can have been no other than poliomyelitis. My great-aunt sustained extensive paralysis and remained a deformed cripple for life. My grandfather sustained a dropped foot, and an adductor paralysis. The date of the occurrence is uncertain, but my grandfather had already displayed an aptitude for cricket, so I do not think that he can have been less than twelve years old. He completed his M.A. degree, and left this country to join the Indian Civil Service at the age of nineteen, and my information does not suggest that his illness occurred during his university career. This narrows the issue to the age of twelve to fifteen years, and, as he was born in 1838, to the years 1850 to 1853 approximately. The tendo calcaneus was divided and a walking calliper applied in 1883. The crippling of the leg did not impair other bodily or mental activities. He served for twenty-five years in the Indian Civil Service, became Commissioner of the Metropolitan Police, and associated with the Special Branch of Scotland Yard in its early days. He was responsible for the security measures in Westminster Abbey at the time of Queen Victoria's Jubilee in 1887, and, owing to the fact that he could not kneel, he was admitted to the private presentation held in connection with the jubilee celebrations. This piece of history is inserted for three reasons. In the first place it serves to confirm that paralysis resulting from poliomyelitis does not necessarily interfere with a full and even brilliant career. Secondly, I have been unable to find an earlier recorded instance of two cases occurring simultaneously in the one household, the earliest noted in the "Bibliography" having been published in 1879(9). Thirdly, a connection by marriage of the family deemed that the simultaneous affliction of a brother and sister in this way was due to the "Evil Eye", thereby causing much distress to my great grand-parents, and that less than a century ago. I take this to mean that the disease was rare, and its nature not understood.

HISTORICAL SURVEY REFERENCES.

- (1) Mitchell, J.K. Tr: A: Am: Physicians, 15: 34,  
1900.
- (2) Hamburger, O. Ugeskr. f. laeger, 73: 1565, 1911.  
Hansen, E. Hosp: Bull: Univ: Maryland, 8: 192,  
1912-13.  
International Committee, "Poliomyelitis", 1932.  
Nörlund, P. "Buried Norsemen at Herjolfsnes".  
Meddleser om Grønland, 67: 1, 1924.
- (3) Hansen, F.C.C. Meddleser om Grønland, 67: 291,  
1924.
- (4) Hippocrates. "The Epidemics". Book I. Third  
Constitution.
- (5) Underwood. "Debility of the Lower Extremity",  
2nd Ed: 1789.
- (6) Heine, J. "Beobachtungen über Lähmungs Zustände  
der unteren Extremitäten und deren  
Behandlung". Stuttgart, Kohler,  
1840.
- (7) Medin, O. "Über eine Epidemie von spinaler  
Kinderlähmung". Verhandl: d. X.  
Internat. med. Kongr. 1890.  
2: Abt. 6, 37, 1891.
- (8) Wickman, I. "Beiträge zur Kenntris der Heine-  
Medinschen Krankheit (Poliomyelitis  
Acuta und verwandter Erkrankungen)  
Berlin, Karger, 1907.
- (9) Packard, F.A. "Acute anterior poliomyelitis  
occurring simultaneously in a  
brother and a sister", J. Nerv. &  
Ment. Dis. 26: 210, 1879.

HISTORICAL SURVEY REFERENCES.

- (1) Mitchell, J.K. Tr: A: Am: Physicians, 15: 34,  
1900.
- (2) Hamburger, O. Ugeskr. f. laeger, 73: 1565, 1911.  
Hansen, E. Hosp: Bull: Univ: Maryland, 8: 192,  
1912-13.  
International Committee, "Poliomyelitis", 1932.  
Nörlund, P. "Buried Norsemen at Herjolfsnes".  
Meddleser om Grønland, 67: 1, 1924.
- (3) Hansen, F.C.C. Meddleser om Grønland, 67: 291,  
1924.
- (4) Hippocrates. "The Epidemics". Book I. Third  
Constitution.
- (5) Underwood. "Debility of the Lower Extremity",  
2nd Ed: 1789.
- (6) Heine, J. "Beobachtungen über Lähmungs Zustände  
der unteren Extremitäten und deren  
Behandlung". Stuttgart, Kohler,  
1840.
- (7) Medin, O. "Über eine Epidemie von spinaler  
Kinderlähmung". Verhandl: d. X.  
Internat. med. Kongr. 1890.  
2: Abt. 6, 37, 1891.
- (8) Wickman, I. "Beiträge zur Kenntris der Heine-  
Medinschen Krankheit (Poliomyelitis  
Acuta und verwandter Erkrankungen)  
Berlin, Karger, 1907.
- (9) Packard, F.A. "Acute anterior poliomyelitis  
occurring simultaneously in a  
brother and a sister", J. Nerv. &  
Ment. Dis. 26: 210, 1879.

### AETIOLOGY.

Observers in the decade prior to the publication of Medin's paper had begun to regard poliomyelitis as a communicable disease. Before the end of the nineteenth century, bacteriological methods were being employed in its investigation. It was not, however, till 1908, the year following the publication of Wickman's paper, that Landsteiner and Popper(1) succeeded in infecting monkeys with material from human cases, and a year later the experimental transmission from animal to animal was achieved.

Flexner and Lewis(2), in 1910, successfully transmitted the disease to rhesus monkeys by instillation into the nose, and demonstrated that the route of spread was by way of the olfactory bulbs to the central nervous system. This observation coloured the whole conception of the disease for the next quarter of a century and more. Spread was deemed to be from nose to nose.

The early workers failed to find any bacterium to blame for the causation of the disease, and concluded that it was one of the filter-passing and ultra-microscopic viruses(3). They were satisfied that it was a living organism. Various experiments with filters of different kinds indicated that the virus was extremely small, and with definite physical and chemical characters.

The current conception of the virus is set out in the following paragraphs (with acknowledgements to van Rooyen and Rhodes).

### Morphology.

No elementary bodies have been described. Inclusion bodies have been described, but have been considered by other observers to be nucleolar extrusions of the host cell. The extremely small size of the virus makes the occurrence of either type of body unlikely.

### Size and Filterability.

Ultrafiltration and ultracentrifuge methods indicate a diameter of approximately  $15 \mu\mu$ .

### Cultivation.

Virus has been demonstrated to survive numerous passages through such substances as monkey plasma, veal broth, and more complicated combinations of material from organic and inorganic sources. Van Rooyen and Rhodes conclude that artificial cultivation of the virus is a matter of considerable difficulty.

### Reaction/



### Reaction to Physical Agents.

The virus is certainly killed by exposure to a temperature of 55°C. for 90 minutes, while it survives a temperature of 0°C. for 40 days.

Dessication in vacuo is survived for a month. At the same time the virus remains viable in water at room temperature in the dark for three months.

Ultra-violet radiations and sunlight are rapidly lethal, and so are supersonic vibrations. On the other hand, X-rays are without effect.

### Reaction to Chemical Agents.

The virus of poliomyelitis displays to perfection the glycerol-resistance of this whole group. It can survive for periods up to eight years in glycerol up to 50% without loss of virulence.

It is killed by most of the normal test chemicals within normal periods. In this connection it is to be noted that it is killed in water by chlorine in a strength of 4 parts per million in 24 hours. Water supplies of the larger undertakings should therefore be safe, but transmission by public supplies remains a theoretical possibility.

### Reaction to Body Fluids and Common Foodstuffs.

Virus can survive the action of the gastric juices for two hours, and is not destroyed by bile.

It remains viable in butter kept at -20°C. for at least three months, and in milk kept at room temperature for a month.

### Types and Strains of Virus.

The groups of viruses which are responsible for human poliomyelitis are related to those causing encephalomyelitis in mice, and the Teschen disease of pigs. It is not improbable that other paralytic diseases of animals are due to allied organisms, amongst these the "loup-ill" of sheep in Scotland is apparently to be numbered. Recently, the clinical similarity of grass sickness in horses to human poliomyelitis has been pointed out. So far, it appears that no claim has been made that the last-named is a virus disease. If, however, it should prove to be, the speculation that a relationship with the virus of human poliomyelitis will be found seems not unreasonable.

Human poliomyelitis is due to two main groups of strains. In the first are placed those normally transmissible/

transmissible to apes, and in the second, those normally transmissible to rodents. For the most part, the two groups are separate, though some simian strains are adaptable to rodent hosts. Amongst the rodent strains is one which was recovered from a dead rodent in a house where a fatal case of poliomyelitis had occurred. In other instances, strains have been recovered from groups of rodents in which they were responsible for more or less disease.

Antigenic studies suggest that all the virus strains show some relationship. Human strains transmissible to apes are more closely related to each other than to human strains transmissible to rodents. The human groups are more closely related to each other than either is to the virus of murine encephalomyelitis, and the virus of Teschen disease of swine is more remotely related still.

The interference phenomenon has been observed where different strains of poliomyelitis virus have been injected simultaneously, or only a short time apart. Similarly, it has been observed that the virus of lymphocytic choriomeningitis can interfere with that of poliomyelitis. The phenomenon is receiving active attention from the experimentalists, and it is possible that a valid preventive measure may be developed.

There seems to be little to choose between the various strains of virus in their ability to produce epidemics, and paralysis. The operative factor is apparently the introduction of a virus to a community which has no acquired resistance to it.

#### The Location of Virus in the Human Subject, Modes of Transmission, Portals of Entry, and Pathways in the Body.

In common with other infectious diseases, the occurrence and spread of the virus of poliomyelitis depends on the route of departure from its host. Consideration must therefore be given to the sites in which it is typically found in a case of the disease.

##### 1. Nervous System.

The virus may be found, post mortem, in any part of the central nervous system. In a series of cases, the relative distribution, and intensity, will be found, broadly speaking, to co-incide with the distribution of paralysis.

Virus has rarely been recovered from the cerebro-spinal fluid of the human.

Similarly/

Similarly, it has seldom, if ever, been found in the peripheral nerves.

The implication of these observations appears to be that the virus can only proliferate when in close contact with neurones, and at the most, it will only appear in other sites while in transit.

## 2. Respiratory System.

Naso-pharyngeal washings, in paralytic, pre-paralytic, and non-paralytic cases usually contain the virus, but it occurs much less regularly in nasal secretions, nasal mucosa, tonsils and adenoids, pharyngeal mucosa, and tracheal secretions. Although recovery of virus has been achieved up to fourteen days after the onset of illness, from most of these locations, it is usual for them to be virus free by the third day of illness.

## 3. Alimentary System.

Saliva shares the low frequency of occurrence normally found in the naso-pharyngeal secretions. Faeces, on the other hand, constantly contain it in all types of case, and it persists in virtually all cases for the first week of the illness. The proportion of faecal convalescent carriers falls steadily to reach zero after twelve weeks. Virus has repeatedly been found in faeces before the onset of clinical symptoms, on one occasion as much as 19 days prior to onset(4).

## 4. Other Situations.

Virus has been recovered from various lymph nodes. Enlargement of many of the groups of nodes has been taken as an indication that there is a stage of lymphatic spread. The view that this hyperplasia is solely to supply lymphocytes has also been put forward.

Virus was recently recovered from the blood in a human case for the first time(5). The previous persistent failure to achieve this is probably the more important observation, indicating that the blood-stream is not normally invaded, and that it is not the route to the central nervous system from the portal of entry.

I have not found any reference to the occurrence of virus in the urine(6).

These findings indicate that the virus can be expected to leave its host in faeces or respiratory droplets. As a natural consequence, entry to the new host can occur only by the mouth or nose. There are, therefore, three main lines of transmission between host and host.



### 1. From Faeces to Mouth and Alimentary Tract, by Contaminated Food or Drink.

The possibilities under this heading are -

- (a) Access of sewage to water supplies.
- (b) Access of contaminated water to food and milk supplies.
- (c) Handling of food and drink by carriers.
- (d) Transfer of virus to food and drink by contaminated flies.

### 2. From Nose to Nose by Droplets.

The possibilities here are -

- (a) Implantation of infected droplets on the mucosa of the respiratory tract, which becomes the portal of entry.
- (b) Contamination of nasal secretions, and their subsequent swallowing, the mode of spread being respiratory, and the portal of entry, alimentary.

### 3. The Contamination of Food or Drink by Droplets.

Two further possibilities present themselves here-

- (a) Access of infected particles to the tonsillar or pharyngeal mucosa in the course of swallowing food, the mode of spread being partly respiratory and partly alimentary, and the portal of entry, respiratory.
- (b) Access of infected particles to the alimentary mucosa by swallowing, the spread being divided between respiratory and alimentary, and the portal of entry, alimentary.

Consideration of the foregoing suggests that the passage common to respiration and alimentation, namely the pharynx, is a likely portal of entry.

The work of Toomey(7) and others, notably Faber and Silverberg(8), bears closely upon this point. Their work is based both upon animal experiment and the study of human post-mortem material. They conclude that virus enters the body by superficial nerve endings. From these it travels along axons to the first neurones, where it multiplies. According to the site of entry the first neurone may be in the olfactory mucosa, the midbrain, in one of the ganglia, such as the gasserian, in a sympathetic ganglion, or, in the case of the parasympathetic, practically in the visceral mucosa. From the first neurone the central nervous system is invaded by the central connections of the peripheral nerve. Once in the central nervous system it spreads far and near, by short and long tracts/

tracts. If this view of spread, from mucosa to the central nervous system by means of nerve fibres, is correct, the virus of poliomyelitis resembles in its behaviour neurotoxins rather than living organisms. This is the mode of spread held to take place in the cases of the toxins of tetanus, rabies, diphtheria, and certain of the snake venoms. Toomey and his fellow workers believe that the relative importance of the various pathways is as follows:-

- (1) The afferent portion of the trigeminal nerve is a very frequent pathway, from the nose, pharynx, and mouth.
- (2) The afferent portions of the glossopharyngeal and vagus nerves, arising in viscera, are fairly frequent pathways, potentially from the nose, mouth, oesophagus, bronchi, stomach, and intestines.
- (3) The afferent portions of the facial, glossopharyngeal, and vagus, associated with taste are occasional pathways from the mouth.
- (4) The sympathetic system provides an occasional pathway from the pharynx, bronchi, oesophagus, and intestines.
- (5) The parasympathetic and olfactory systems are not involved.

The general inference to be drawn from this work is still that the portions of the body common to respiratory and alimentary systems are more likely to be portals of entry than the lower alimentary tract. Further, implantation of virus at any one point in the mucosa may lead to involvement of the grey matter over a wide area, capable of marked variation from case to case. The distribution of paralysis in any one case, therefore, throws no light on the mode of transmission, portal of entry, or pathway of spread. This generalisation must be qualified in respect of the known fact that tonsillectomy tends to pre-dispose to bulbar paralysis. In this case the portal of entry and pathway of infection are only too obvious. The available evidence suggests, in fact, that all the modes of transmission, portals of entry, and pathways of spread so far mentioned require serious consideration, and that, therefore, measures of control should cover them.

#### The Vehicle of Transmission of Infection.

It is possible for infection to pass in a variety of ways, thus -

- (1) From case to case by direct contact.
- (2) From carrier to case by direct contact, in this event the carrier may be -
  - (a) Already incubating the disease.
  - (b) A convalescent carrier.
  - (c) A carrier giving no history of morbidity nor of contact.

(3)/

- (3) From case or carrier to food or drink by droplets.
- (4) From case or carrier to food or drink by faecal contamination.

If transmission were commonly by droplets, considerable outbreaks in relatively closed communities, such as households, schools, factories or service establishments might be expected. Second cases in families are far from unknown, but are rather unusual. In the same way, school outbreaks by classes, so common in the exanthemata, are unusual.

Similarly, outbreaks clearly traceable to food and drink are unusual. In a number of instances, however, there have been grounds for suspecting that infection could have been conveyed by milk. Thus, at Broadstairs, 42 cases occurred between 10th and 17th October 1926, and a further 20 cases up till 29th October(9). The great majority of cases, especially the early ones, occurred in residential schools with a common milk supply, and few cases occurred amongst the townsfolk.

While the explosiveness of the outbreak, and the restricted population affected, coupled with the common milk supply, is enough to raise suspicion, the fact that the incidence in the town was low, although the same milk was sold there, suggests that this outbreak was not due to milk. Other outbreaks ascribed to milk tend to have the same characteristics, that is, there is always some indication of spread by other means, or that the chain of reasoning is defective in some link.

Water as a vehicle has received its share of attention. Various observers(10) have drawn tables or graphs to show that the occurrence of cases in the outbreak described was associated with the flow of water, the nature and source of the water supply, the quantity of water consumed daily per head of population, or the method of removal of waste water. No rule of universal application has been deduced from these studies. It is perhaps worth pointing out that in Great Britain, especially in England, water supplies have often to be taken from potentially polluted streams, and thereafter purified. The recommendations contained in the Eighth Report of the Royal Commission on Sewage Disposal are, however, carefully observed. In the United States, much attention is paid to purification, but little to the proper disposal of effluent along the lines of our Royal Commission's findings, water-borne disease is as a result substantially commoner there, and it may be that this is a factor in the continued high incidence of poliomyelitis in that country.

The/



The foods which have received particular attention are those which display a seasonal incidence similar to that of the disease, namely the "kindly fruits of the earth"(11). Here again, the evidence is not wholly convincing. These fruits are not available in some of the areas where poliomyelitis has occurred. The mode of action may be that surfeit produces gastro-enteritis, and that this condition predisposes to the entry of the virus if it happens to be present.

Van Rooyen and Kirk(12) are very eloquent concerning the danger of infection with poliomyelitis, as well as many other diseases, through the consumption of raw vegetables and fruit in India and neighbouring oriental lands. The danger lies in the manuring of these crops with human excrement. During the Siege of Malta, there was difficulty in obtaining manure for crops, and, in spite of all orders to the contrary, it appears that human excrement was used. Whether this was the cause of the severe outbreak of poliomyelitis, which occurred shortly afterwards, cannot be proved, but can be suspected.

Arthropoda have received their due share of attention. As the virus is not blood-borne, no consideration need be given to the blood-sucking arthropods. The possibility of mechanical transmission by flies is, however, very real, and virus has been recovered from house-flies. Infection of flies seems to imply access to faeces, sewage or contaminated water courses. On the whole, the hygienic failures hereby implied are more likely in the country than in the town, and it is in just the direction of preventing the occurrence of these failures that hygiene has advanced in the last century, similarly it is in the last century that the disease has advanced. For this reason it does not seem probable that the fly ranks high as an important fundamental vehicle of spread.

To summarize, therefore, it is known that some outbreaks have given an epidemiological picture resembling that of spread by water, milk, food, drink, and flies in other diseases. It is undoubtedly possible that these outbreaks could have resulted from spread by the vehicle in question. There remains, however, no vehicle of spread universally present other than the human subject.



## EPIDEMIOLOGY.

### Distribution of Cases.

No part of the world is exempt from the disease. At the same time, there is some connection between climate and the incidence of severe cases. In the Northern States of the U.S.A., in Canada, in Sweden and other countries of Northern Europe, in Australia, New Zealand, and South Africa, the disease may be said to have its epidemic home.

There is definite association in these lands with warm weather, though not with its onset. Where a continental type of climate is found, alternating between winter frosts and summer heats, the prevalence tends to be high. Especially is this true of the North American Continent, and of Scandinavia, but it must be remembered that Russia, Siberia, and North China, where the same type of climate prevails, are relatively free from poliomyelitis. This may mean merely that they have not yet had their turn, but it might also result from differences in hygienic standards, or many other causes not immediately apparent.

In the Southern Hemisphere, the disease is well known in Australia and New Zealand, where the incidence in 1925 reached 100 per 100,000, against that in England and Wales in 1947 of 18 per 100,000. There seems to be no climatic reason against a very much higher rate in this country.

### Distribution of Epidemics.

Severe epidemics tend to be a feature of the Northern States of the U.S.A., rather than of the Southern. Epidemicity has been a usual occurrence in Sweden, Norway, Denmark, other European countries, and lands in the Southern Hemisphere. A steady annual incidence has been noted in the Southern U.S.A., and in neighbouring tropical parts of America. There is evidence of endemicity in the lands bordering the Mediterranean, the Middle East, and India. Troops from the United Kingdom, and the U.S.A. sent to these areas during the recent war suffered higher incidences than similar formations in their home-countries. Their incidence was that of their age-groups in an unsalted community, while that of the local populace was that of a salted community. In Malta this finding was particularly clear. The explanation appears to be that the troops encountered a strain of virus to which they had no immunity, and therefore different from that of their home-countries, but to which the local populace was resistant.

Epidemics do not occur in all parts of the U.S.A. in the same year, but the seasonal prevalence for the whole country has remained fairly steady at 5 to 10 per 100,000, since 1916. The annual prevalence is made up to the sum of a number of local epidemics. The following table illustrates the point.

(Rates per 100,000)	<u>1924</u>	<u>1926</u>	<u>1929</u>	<u>1930</u>
National Rate (approx.)	5	3	3	7
New York City	6.0	2.0	1.0	1.1
Syracuse	71.0	35.0	12.0	74.0
Rochester	2.0	3.0	10.0	21.0
Buffalo	6.0	26.0	18.0	13.1

These figures are taken from tables published in the International Committee's work. They demonstrate that the local incidence may vary much more widely than the national.

In Great Britain, the population is one third that of the U.S.A., living in about one fortieth of the area. Here it seems improbable that an outbreak could assume epidemic proportions in a substantial part of the country, leaving other parts with normal inter-epidemic incidence, and that this process could continue year by year so that the annual incidence remained steady.

#### Cause of Epidemics.

The main cause of any epidemic must always be the accumulation of persons susceptible to the infecting agent. In respect of poliomyelitis this could result from prolonged low incidence, change in the nature of the virus, by the introduction of a new type or mutation of the old, or by changes in the environment of the community. This in its turn might result from unusual weather, or an altered standard of nutrition, housing, or sanitation.

In the United States of America, evidence that outbreaks are more likely in years of exceptional meteorological conditions has not been found. In Great Britain, however, there is some evidence that a hot summer favours a higher prevalence. This will be further considered in connection with the British outbreak of 1947.

#### Incidence.

##### Seasonal Incidence.

Seasonal incidence is associated with the return of warm weather, and the seasonal decline of incidence with the return of colder conditions.

It/



It has been pointed out that the seasonal incidence is that of enteric and this fact has been used as evidence that the mode of infection is by ingestion. This should be accepted with caution. Enteric has been virtually banished from Great Britain as a disease with a seasonal incidence. This has been done by securing safe water supplies, and by the sanitary disposal of sewage. Outbreaks of enteric nowadays depend on the existence and activity of carriers, and on their access in an infective state to food and water. Examples of the access of carriers to water are the Croydon outbreak of 1938, and the Greenock outbreak of 1948. The contamination of the Croydon water by a workman who was a carrier could have happened on any day in the year but the results would have been the same. The contamination of the stream associated with the Greenock outbreak could have happened on any day in the year, the factor determining the seasonal incidence was the suitability of the weather for picnicking. Examples of the access of carriers to food are the Hawick outbreak of 1938, and the Inverness outbreak of 1941. These two outbreaks depended on the contamination of bakery produce, and again this could have occurred on any day of the year. Before it is concluded that the seasonal incidence of poliomyelitis has anything in common with that of enteric, it should be demonstrated that measures which have been successful in preventing the spread of enteric should interfere with the spread of poliomyelitis. This is clearly not the case, rather the reverse, for poliomyelitis has tended to increase contemporaneously with the hygienic improvements which have so profoundly altered the incidence of enteric. The alternative conclusions appear to be that the virus changes with the seasons, or that its host does. It may be that the virus requires the warmer weather for full physiological development, that at this time it can be transmitted more readily, and that therefore exaltation of virulence by passage occurs most readily at this season of the year. If there is a connection between warm weather and the virus, it seems likely that whether this be incidental to metabolism or to the opportunity of passage, a late summer prevalence of the disease would result, and that if both factors operate, this effect would be more pronounced. In the same way, the decline in the incidence as the colder weather sets in would result from depressed metabolism on the part of the virus occurring at the same time as the decline from maximum virulence due to excessive passages. The observations of Smith, et al:(1) on the outbreak in Buffalo and Erie County in 1944 bear on this point. They write:-

"Patient 1, (a 4 year old child), developed paralytic poliomyelitis at the end of an eleven week period during which 8 of his 9 associates had illnesses with/

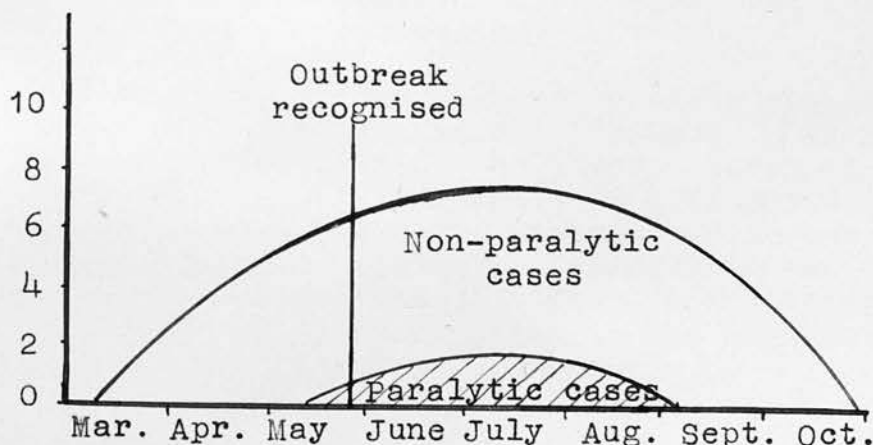
with incubation periods of approximately two weeks. At least one of these illnesses was highly suggestive of so-called abortive poliomyelitis..... Circumstantial evidence would suggest that these illnesses represented mild forms of poliomyelitis and were followed by definite paralytic symptoms in the 4 year old child. The incubation period of approximately two weeks is distinctly different from that of common respiratory diseases with which poliomyelitis is most likely to be confused. Judging from this study one would postulate that the poliomyelitis virus had been active in the community for at least eleven weeks prior to the first paralytic case of the disease."

It should be noted that the authors' reconstruction of events implies five passages of the virus through human hosts in those eleven weeks.

Two other cases of paralytic poliomyelitis occurred on 24th May and 28th May respectively. The former was associated with four cases of minor illness suggestive of abortive poliomyelitis. The latter occurred simultaneously with two cases in the family and followed three other cases of illness in associates, resembling non-paralytic poliomyelitis.

The last cases of 1943 occurred 25 miles away from those noted in 1944, and five and a half months before the first recognised case of 1944. The authors' survey covers 11 of those 24 weeks, and it seems not unreasonable to bridge the gap of 13 weeks with supposed abortive cases of poliomyelitis, masquerading as infections of the upper respiratory tract, and spreading centrifugally along the normal channels of human intercourse.

The authors were fortunate in that the records of absence from school, with causes ascertained by the school nurse, were readily available. With this and other information they were able to draw a chart, which may be schematically rendered thus:-



No reliable figures were obtained concerning concurrent minor illness in the community, nor concerning its bearing on the outbreak. They gained the impression, however, that "such illnesses were very prevalent both before and during the epidemic. The often quoted estimates of 10 to 40 abortive cases for every one in which paralysis develops would not seem unreasonable in the light of our experience." They were, however, unable to determine that there was any difference between the incidence of these diseases in 1944 and previous years.

The conclusions to be drawn from these observations appear to be as follows:-

- (1) It is a fact that an outbreak of paralytic poliomyelitis is preceded, accompanied and followed by an increased prevalence of non-paralytic cases. The severity of the non-paralytic cases varies from those with all features of the disease, short of paralysis, to cases of apparent coryza, influenza, or gastro-intestinal disorder.
- (2) It is highly probable that there is no season of the year in which there is no prevalence. On the contrary, there is a strong suggestion that the virus persists in the neighbourhood of the last reported case in one year, giving rise to cases of minor ailments, the true nature of which pass unrecognised.
- (3) There is considerable evidence that the incubation period, at any rate in connection with non-paralytic cases, is about two weeks in length.
- (4) The onset of warmer weather is in some way necessary for the development of virulence. This condition may be required for the proper multiplication of virus, with paralysis developing from a heavy dose of virus in a susceptible subject. Or, the warmer weather may facilitate the passage of virus, perhaps by prolonging its viability, or by providing insect vectors. Or the warmth may of itself exalt the virulence of the virus or depress the resistance of the host, this lowered resistance perhaps being confined to the central nervous system.
- (5) The onset of colder weather, broadly speaking, coincides with the decline of virulence. It may depress the metabolism of the virus, thereby reducing the dose which can be transmitted. It may increase the resistance of the host, in toto, or in respect of the central nervous system. Its arrival may coincide with the natural decline of virulence following exaltation. Or the diminution in the numbers of insect vectors may be effective.



### Age Incidence.

Heine described the disease as affecting the young child, to the virtual exclusion of the older child or adult, except in exceptional cases. This infantile incidence is quite clear in the case of other early writers such as Badham. The infantile incidence was found in recent outbreaks in Malta(2), Mauritius and South Africa. From the time of the first great outbreaks in Scandinavia and the United States of America in the first and second decades of this century, the age incidence in these countries, and in Australia, has shifted to the schoolchild. This altered incidence has affected urban populations more than rural, and is more pronounced in the cooler countries, or parts thereof, than in those nearer the tropics.

The conclusion generally drawn from this finding is that the incidence is in the young child where the virus is endemic, all other age groups having acquired immunity. Where, however, environment is especially good, and hygienic measures especially complete, the age incidence rises, the child being especially well protected against infection until he begins to go about, thus reaching school age without acquiring immunity.

In 1932, while on my way to Southern Rhodesia, I visited the island of St. Helena, shortly after an epidemic of measles. I learned that it was the first outbreak for many years. Of the five thousand inhabitants, three thousand had contracted the disease. Those who escaped were all in the older age-groups, who had suffered in a previous outbreak. This is a fair example of the way in which an "unsalted" community, or portion of a community, reacts to a virus of high infectivity when first exposed.

In 1945 and 1946, as already noted, the same island community sustained its first considerable outbreak of poliomyelitis. The reports(3) show that the incidence in the age-group 10 years to 25 years predominated, and that the young child was relatively immune.

A fair deduction from the foregoing appears to be that in an entirely "unsalted" community, all ages are open to attack, as in measles, but that the virus has an especial predilection for the adolescent and young adult.

Occasionally it happens that two communities, of differing degrees of immunity to the local virus, are exposed under essentially similar conditions. The outbreak in Malta, already mentioned, may be cited as a classical example(2). Amongst the civilian population of that gallant island there were 426 cases. Of these, 80% were under the age of five years, the two year-old and/

and three year-old age groups being chiefly involved. Only 4 Maltese adults were affected. In the garrison there were 57 cases, naturally all in adults. At the time in question the civil populace numbered approximately 270,000, of whom approximately 170,000 would be adults. At the most, the garrison numbered 40,000. This gives relative incidences amongst adults of 2.1 per 100,000 in the civil populace, and 142.5 per 100,000 in the garrison. While these figures are not meticulously accurate, they are sufficiently so for the comparison to be made.

I am indebted to Dr. A.C. MacDonald, of Elgin, for much of the foregoing information. He was a colonel in the Royal Army Medical Corps, and stationed in Malta throughout the siege. He has drawn my attention to an interesting facet of the garrison outbreak, namely that the incidence amongst Royal Air Force personnel was both relatively and absolutely greater than amongst those of the Royal Navy and the Army. This applied equally to air crews and ground staffs. I record this without attempting to explain it.

So far, in this section, I have proceeded on the assumption that there are different strains of virus, and have accounted for the variations in age incidences on this assumption. If the data is examined from another view-point it is clear that it forms strong evidence in favour of varying strains, and varying states of immunity to them. Thus the American G.I., presumably immune in some degree to transatlantic strains, proved to be no more immune, in the Near and Middle East, than his British comrade in arms. Both were much more susceptible than the local inhabitants. Both demonstrated incidences greater than those of the inhabitants. The experience of both can only be explained on the basis of lack of immunity to a local virus strain differing from that of their respective homelands. The argument can be carried a step further. It is fair to assume that the American was more likely to have acquired a degree of immunity than the Briton, in view of the greater prevalence in the United States for many years past. Nevertheless, when the two, one "immunised" and the other not, are transplanted to a field of unexperienced virus potential, both succumb, thereby showing that the experience of the one, and the inexperience of the other made no difference.

#### Sex Incidence.

There is little to discuss in this connection beyond recording the facts. In general, there are approximately 4 male cases for every 3 female cases. There is some evidence that after the age of 20 years this ratio is reversed, and that among adults, female cases preponderate.

Racial/

### Racial Incidence.

There is evidence of variation in racial susceptibility, though none of racial immunity. Broadly speaking, the European, wherever domiciled, is more susceptible than the African, Indian, Chinese, Maltese, Filipino and Japanese. How far this is intrinsically racial, and how far due to differing social habits is not apparent. Hitherto, the European has probably developed community hygiene to a higher degree than the other races. The suggestion that increased susceptibility to poliomyelitis is the result of successful social hygiene has already been noted.

### Infectivity.

The infectivity of poliomyelitis may be judged by comparing its incidence with that of the common infectious diseases, measles, whooping-cough and scarlet fever. This has been done in the following table, the communities selected being Edinburgh and Glasgow in the year 1947(4). There is nothing to suggest that the experience of these two cities was unusual in that year.

Table of Notifications of Measles, Whooping-cough, Scarlet Fever, and Poliomyelitis in Edinburgh and Glasgow, 1947.

	<u>Edinburgh</u>	<u>Glasgow</u>
Measles.		
1st Quarter.	828	533
2nd Quarter.	276	630
3rd Quarter.	178	349
4th Quarter.	121	2609
Total for year.	1403	4121
Whooping-cough.		
1st Quarter.	311	3609
2nd Quarter.	287	1558
3rd Quarter.	142	161
4th Quarter.	49	179
Total for year.	789	5507
Scarlet Fever.		
1st Quarter.	139	1273
2nd Quarter.	98	630
3rd Quarter.	70	475
4th Quarter.	111	1291
Total for year.	418	3669
Poliomyelitis.		
1st Quarter.	1	2
2nd Quarter.	2	7
3rd Quarter.	87	391
4th Quarter.	49	85
Total for year.	139	485



The clear-cut seasonal incidence of poliomyelitis makes comparison through the whole year worthless, but if the third quarter be taken, it is seen that in Edinburgh, in that quarter, its incidence was exceeded by that of measles and whooping cough, and slightly exceeded that of scarlet fever. In all three diseases used for comparison the quarterly figure was, if not the lowest, the second lowest for the year. In Glasgow, the figure for poliomyelitis exceeded that for measles in the third quarter, by about 10%, but the figure for the latter was by far the least for any quarter in the year. In respect of whooping-cough, the quarterly figure was more than doubled, though it is clear that there was an approach to epidemic prevalence in the two previous quarters. The quarterly figure for scarlet fever, although the lowest for the year, exceeded that for poliomyelitis by nearly 20%. Broadly speaking, therefore, it may be said that the maximum incidence of poliomyelitis in an epidemic year is equal to the minimum annual incidence of the common infectious diseases. It is probable, however, that if every abortive and sub-clinical case were identified, the total morbidity would be very much nearer that, say of measles, which attacks every fully susceptible person who is fully exposed.

In another respect poliomyelitis differs from the other infectious diseases, and that is in the relative rarity of second cases in the same household, residential school, or other similar semi-closed community. When the writer had measles, a brother introduced the infection into the household, and the remaining four children succumbed to the infection on the eleventh day. Such an occurrence is common with measles, and uncommon with poliomyelitis. Horstmann and Paul quote from the Yale Poliomyelitis Study Unit an extreme instance of familial involvement(5). In a family of eight children aged from 3 years to 10½ years, there were three paralytic cases, one being fatal, two further cases with evidence of meningeal irritation, but in which no paralysis developed, and three cases with minor symptoms, which were presumed to be abortive.

Where hospital staff, particularly nurses, are concerned, the risk of contracting the disease as a hazard of occupation appears to be negligible, thus confirming that the case once recognised is hardly to be considered as a source of infection. This finding suggests in its turn that it is the missed case, or the carrier that spreads infection.

Poliomyelitis shares with meningococcal meningitis and encephalitis lethargica a certain appearance of capriciousness in its choice of victims. In fact the histories of these three diseases have much in common.

Two/



Two hundred years ago, none of them was recognised as an entity. In the last two centuries all three have developed first a prevalence in the community, followed by an epidemic tendency, and finally a very greatly increased incidence. It is customary nowadays to point to the increased nervous strain resulting from the pace of modern life. Presumably this could be an aetiological factor, but it is only reasonable to point out that, in the nineteenth century the increased incidence was well under way, and that, in the words of H.G. Wells, it was an age "of such widely diffused plenty, cheapness and freedom as no man living will ever see again".

It is possible that the key to the proper understanding of poliomyelitis is to be found in its epidemiological similarity to meningococcal meningitis. In this disease, it has been demonstrated that the carrier state is constantly found in the community, to the extent of five per cent on the average at any one time(6). The vast majority of these carriers never suffer from more than a passing naso-pharyngeal catarrh, and this carrier rate is associated with the occasional sporadic occurrence of cases of meningitis. Under certain circumstances the carrier rate may rise, and it has been found that a rate of twenty per cent precedes an outbreak of epidemic severity. Such outbreaks and carrier rates have come to be associated with the overcrowding in service establishments, such as barracks, hutments, or billets, and it has been supposed that faulty ventilation was the operative factor, permitting a high carrier rate with its inevitable trail of cases of meningitis. Recently, however, attention has been paid to the fact that service personnel in such surroundings are for the most part strenuously engaged in military training, and the suggestion has been put forward that the physiological reactions of the nervous system are changed, leading to increased susceptibility to meningococcal invasion(7). It appears unlikely that either factor is the only one operative, and that the incidence is a resultant of altered conditions in both the organism and its host. If poliomyelitis is regarded in the light of the foregoing, it must be assumed that the virus is continually present in some members of the community, who suffer no, or minimal effects, and that some combination of changes in its and its host's environment and physiological reactions produces epidemic prevalence. If this be the case then those who sustain recognisable poliomyelitis are the battle casualties resulting from an invasion of pandemic proportions.

To summarise, therefore, poliomyelitis is a disease which, judged by the number of recognised cases it produces, is of low infectivity, but judged by the number of infections which probably occur without giving rise to any morbidity, it is an infection of high infectivity, but low potential for causing harm.

Delayed Infectivity.

My attention has been drawn to the occurrence of second cases of poliomyelitis after many years in the same households in the Island of Skye. This would appear to suggest that the virus may, under certain conditions, persist locally for much longer than has hitherto been demonstrated(6).

Mortality.

Death occurs on account of bulbar paralysis, involvement of the cervical cord causing diaphragmatic paralysis, and, if the condition be accepted as true poliomyelitis, polioencephalitis. The death-rate will therefore vary with the incidence of bulbar and diaphragmatic palsies. The authorities give 20% as an average figure, with extremes of 5% to 50%. I note that recorded incidence of paralysis varies from one outbreak to the next, and presume that, to some extent, the mortality rate varies inversely with the reporting of non-paralytic cases.

EPIDEMIOLOGY REFERENCES.

- (1) Smith, M.L., "A Study of the Origin of an  
 Bridge, E.M., Epidemic of Poliomyelitis".  
 Underwood, H.E. J.A.M.A. 129: 17, 1945.  
 & Dale, G.E.
- (2) Agius, T. et. al: Brit: Med: J: 1: 759, 1945.  
 Bernard, A.V. Brit: Med: J: 2: 26, 1945.  
 Bernstein, H.G.G., Brit: Med: J: 1: 763, 1945.  
 Clark, J.M.P. &  
 Tunbridge, R.E.  
 Debono, J.E. Brit: Med: J: 2: 61, 1945.  
 Seddon, J.H., Lancet. 2: 707, 1946.  
 Hawes, E.I.B., &  
 Raffray, J.R.  
 Morana, J. J: Roy: Inst: P: H: & Hyg:  
 8: 181, 1945.
- (3) Colonial Office Personal communication to  
 Report. Rhodes, A.J. quoted in Bull:  
 Hyg: 22: 6, 1947.
- (4) Department of Weekly Bulletins of Infectious  
 Health for Disease.  
 Scotland.
- (5) Horstmann, D.M. J.A.M.A. 135: 11, 1947.  
 & Paul, J.R.
- (6) Johnston, E. Personal communication.

## PATHOLOGY.

### The Central Nervous System.

Heine(1), later supported by Charcot(2), considered that the essential lesion of poliomyelitis was necrosis of the motor cells in the anterior horn of the spinal cord. This is the view held to-day by the orthodox. The issue was, however, complicated by disputes concerning the primary involvement of neurones or neuroglia, the role of the vascular system in the causation of lesions, and the mode of spread to, and in the central nervous system. From the work of pathologists and others, over a period of half a century, the present day view has crystallised.

The mode of entry and route to the central nervous system, though properly part of the pathology of the disease, have already been considered in the section devoted to aetiology, as they throw light on epidemiological problems, and the habits of the virus. It is therefore sufficient to re-state here that the virus reaches the central nervous system by travelling along the axon of an afferent neurone from one or other of the mucous surfaces of the body.

Having reached the central nervous system, the virus is found to act as an obligatory intracellular parasite of neurones. The nature of the damage is degeneration or necrosis. The extent of damage, and its severity therefore depend on the number of neurones involved, and the severity of their involvement. Where the severity is intense, acute necrosis takes place. If the intensity is insufficient to cause necrosis, varying degrees of interference with cell structure and function occur, and recovery will tend to take place.

Where cell death takes place, amoeboid microglial cells, and polymorphonuclear leucocytes rapidly appear to exercise their phagocytic functions. They are accompanied by the exudation of fluid, and fill the perivascular lymph spaces in the cord, giving rise to the appearance of cuffing, and may overflow into the cerebrospinal fluid.

Where cell death does not take place, the microglial cells are absent, but in other respects the sequence of events is the same.

The overflow of cells into the cerebrospinal fluid prompted the view that the virus spread to the meninges by the blood stream or lymphatics, gave rise to meningitis, and then invaded the brain and cord from without inwards. If an experimentally infected animal is killed at the first sign of clinical disease, it is found that the only lesions are in the neurones/



neurones(3). The exudation of fluid and cells occurs after this neuronal involvement, and is wholly reactionary in nature. By the time that a human case dies from natural causes the whole process of exudation and phagocytosis is well under way, and perivascular cuffing is present.

In the neurone itself, the finding appears to be that necrosis may take place within the first twenty-four hours after the onset of symptoms. If, however, this period is survived, recovery will almost certainly take place. The events usually seen in such cases include chromatolysis, nuclear shrinkage, nucleolar extrusion, uneven staining of cytoplasm with reticulation and vacuolation, granular disintegration with loss of the intracellular fibrils. Occasionally the cell and nucleus swell up and become very pale. In either case, the attached myelin sheath may show some distension and later may become fragmented.

These changes are most intense in the anterior horn cells of the lumbar and cervical enlargements of the cord. They are to be found, however, in the thoracic cord, brainstem, basal ganglia, cerebellum and motor cortex. Necrotic lesions within the skull are said by some observers to be less frequent than those which are reversible.

As the attack on neurones dies down, the acute phase of the illness comes to an end. The removal of necrotic tissue is followed by the cessation of phagocytosis, reabsorption of fluid and the disappearance of perivascular cuffing. Where neurones have been destroyed there is an excess of glial cells. Changes in the myelin sheaths of damaged but surviving neurones may persist for some time. The bulk of clearing up is achieved within two months of the onset of the illness. The restoration of function in damaged neurones is generally considered to be complete within six months to two years, but there is some evidence to the effect that recovery can take place at a much later date, and this point will receive further consideration later.

#### The Cerebrospinal Fluid.

From the beginning of the present century, changes in the cerebrospinal fluid have been noted, and the experience of several decades indicates the usefulness of its examination as an aid to diagnosis. The commonest findings are outlined in the following paragraphs.

#### Pleocytosis.

The fluid contains a moderate increase in the number of cells, due principally to lymphocytosis, and in/

in a much smaller degree to increase of granulocytes. In most cases the cell count is within the range of 10 to 300 per c.mm. Higher counts are not unknown, but are unusual. Normal counts have been recorded in paralytic and even fatal cases with no great infrequency. The pleocytosis is of short duration, the findings having usually returned to normal within fourteen days of the onset of symptoms.

#### Increase in Protein Content.

The protein content of the cerebrospinal fluid rises above the normal limit of 40 mgm: per cent. later in the illness than the pleocytosis, so that its figure is rising as the cell count falls. The protein content remains raised for a few days longer than the duration of the pleocytosis. Normal protein contents may be found in severe cases, in the same way that normal cell counts are so found.

#### Other Findings.

The pressure of the fluid is commonly raised, and this may be found when there is no other abnormality.

Other chemical constituents are present in normal quantities, and their estimation is only necessary for purposes of differential diagnosis.

The colloidal gold test is inconclusive.

The colour and clarity of the fluid are virtually normal, but on standing a fine clot may form.

#### Significance.

The significance of changes in the cerebrospinal fluid is still a matter of controversy. In some areas, a diagnosis of poliomyelitis is not considered unless there is a pleocytosis of not less than 10 cells per c.mm. Other observers, while admitting the value and importance of examination of the fluid, refuse to be bound by laboratory findings, and are prepared to make a diagnosis of poliomyelitis on clinical grounds, if necessary without laboratory confirmation. Amongst these latter is Jensen(4), who noted that, in the Danish outbreak of 1934, there was a general agreement between the amount of the pleocytosis and the severity of the disease, but at the same time there was a substantial proportion of cases in all grades of severity - even fatal - in which normal fluids was found.

#### Changes in the Muscles.

The changes in the muscles depend on the degree of destruction of neurones. Substantial loss of innervation/

innervation leads to muscular atrophy. Some of the early pathological students of the disease failed to find lesions in the central nervous system, and considered that it resulted from primary fatty degeneration of the muscle fibres. A French pathologist(5) employed the word "graisseuse" to describe the change. It seems probable that these early students had little opportunity of studying pathological material from recent cases, and that they were almost entirely dependent on the examination of material from patients long recovered from the acute stages. It is interesting to see in the Kenny concept of the disease(6), a return to the idea that the lesion is located in muscles, fascia or skin, though not in the muscles the orthodox deem to be paralysed, but in their antagonists.

#### Changes in the Electrical Reactions of Muscle.

The changes in the electrical reactions of the affected muscles are those normally encountered in cases of lower motor-neurone paralysis. They vary according to the severity of the nervous involvement, and to the time that has elapsed since the appearance of paralysis. They are therefore of great value in prognosis.

In normal muscle, there is tetanic contraction on faradic stimulation of the motor point, and single contractions on making and breaking a galvanic current applied at the same point, the kathodal twitch being greater than the anodal.

In partially affected muscles the response to faradic stimulation is slight or absent, and to galvanic present but sluggish. If the stimuli are applied direct to the nerve, there may be little or no reaction. In such cases a substantial degree of recovery is likely, and complete recovery possible.

The typical reaction of degeneration is found in grossly or totally affected muscles. This consists of absence of response to faradic stimulation, worm-like response to galvanic, with greater response to the anodal current. Or, in the very extensively and completely involved muscles, there may be no response at all. The prognosis where there is typical reaction of degeneration or no reaction is bad.

Chronaxie ascertainment may serve a similar prognostic purpose. By this means a measurement of the excitability of the muscle may be made. Immediately after the death of a neurone, or its separation from its axon and muscle fibre, the chronaxie remains unchanged. In the affected nerve it rapidly rises and then disappears. In the affected muscle/



muscle it rises much more slowly, and is to be found after that in the nerve has been absent for two weeks.

### Other Organs and Systems.

#### The Blood.

The increase in granulocytes normally found in acute infective conditions occurs in poliomyelitis, and the count may reach 30,000 per c.mm.

#### Lymphoid Tissue.

Enlargement of the spleen, of the Peyer's patches in the intestines, and of lymph nodes has been noted. These changes do not occur constantly, and their significance is not clear. The changes in the Peyer's patches have been considered to indicate an alimentary portal of entry.

#### Immunity.

In a "salted" population, the whole weight of attack is on the young child. In an "unsalted" population, part of the weight of the attack shifts to the older age-groups, but not all. In other words, some of the immunity of the older people in the former populace results from exposure to infection.

Various tests have been used to determine the state of individual immunity, and of these neutralisation of the virus by serum has been most popular(7). The result of these investigations has been to show that antibody is present in human sera from all parts of the world, and all races, in titres similar to those for other infectious diseases. The child at birth has its mother's antibodies, and in the maternal quantities. By the age of one to five years, the antibody content is at its lowest ebb, and thereafter rises steadily with age. Some observers have found that antibody is present in its highest titre in the sera of convalescent paralytic cases, and this has no doubt been the basis of its therapeutic use(8). The finding has not, however, been universally confirmed(9).

The occurrence of antibody has also been attributed to chance, to other antigenic agents, and to changes occurring in the serum with age.

One view is that exposure to virus leads to a degree of invasion. Some of the virus causes alterations in the serum, but this does not in any way indicate immunity, which is of nervous tissue, and not generalised. There is widespread infection in the community, many persons are invaded, but only the few whose nervous systems are immunologically, perhaps physiologically/



physiologically, susceptible show signs of paralysis(10). There is confirmatory experimental and other evidence that absence of suprarenal cortical substance predisposes to paralysis(11), that female sex hormones tend to protect from paralysis(12), and that the incidence of paralysis in the human varies according to the blood group(13). These are all physiological factors.

PATHOLOGY REFERENCES.

- (1) Heine, "Beobachtungen über Lähmungs-  
stände der unteren Extremitäten  
und deren Behandlung".  
Stuttgart, Kohler, 1840.
- (2) Charcot, J-M., Arch: de physiol: norm: et  
& Joffroy, A. path: 3: 134, 1870.  
Compt: rend: Soc: de Biol:  
1: 312, 1870.
- (3) Fairbrother, R.W. J: Path: & Bact: 33: 17.  
and Hurst, E.W.
- (4) Jensen, C. Poliomyelitis Epidemic in Den-  
mark in 1934. Proc: Roy: Soc:  
Med: 28: 1007, 1935.
- (5) Broca, P. Bull: Soc: Anat: de Paris.  
26: 381, 1851.
- (6) Kenny, E. New York State J: Med:  
42: 1645, 1942.
- (7) Levaditi, C. & La poliomyelite experimentale.  
Landsteiner, K. Compt: rend: Soc: de Biol:  
68: 311, 1910.  
  
Netter, A. & Compt: rend: Soc: de Biol:  
Levaditi, C. 68: 617, 1910.
- (8) Flexner, S. & J.A.M.A. 54: 1780, 1910.  
Lewis, P.A.
- (9) Shaughnessy, H.J., California & West: Med:  
Harmon, P.H., & 33: 754, 1930.  
Gordon, F.B.
- (10) Müller, H. Monatschr: f: Kinderk:  
87: 263, 1941.
- (11) "Poliomyelitis", International Committee.
- (12) "Poliomyelitis", International Committee.
- (13) Grooten, O. & Compt: rend: Soc: de Biol:  
Kossovitch, N. 105: 428, 1930.

### CLINICAL FEATURES.

The characteristic feature of poliomyelitis anterior acuta is the appearance of paralysis of voluntary muscle. This is flaccid in nature, and once established is virtually irreversible. Residual paralysis is liable to lead to deformity. On all counts, therefore, the paralysis is an occurrence to be feared owing to the suffering and loss of economic independence it may impose on its victims. Fortunately, it is not every case in which paralysis occurs, nor does every paralytic case remain without improvement. Early recognition of the disease is essential, not only to the proper management of cases, but also to the institution of preventive measures. Consideration will therefore be given to factors likely to predispose to infection and paralysis.

#### Predisposition to Infection and Paralysis.

The virus is widely prevalent in epidemic periods, but the infectivity of the disease is low. Endocrine disturbance(1), lack of vitamins(2), and malnutrition have been held responsible for this uneven and apparently unpredictable selection of victims, but without convincing proof. More recently, fatigue has been blamed, as a predisposing factor in both morbidity and paralysis. The observations of Russell(3) and Hargreaves(4) confirm that "Physical activity of any kind during the pre-paralytic stage increases the danger of severe paralysis. Complete rest in bed during the whole of the pre-paralytic stage seems to protect the patient from severe paralysis". In his later paper Russell concludes that "severe physical activity at this (the pre-paralytic) stage is almost suicidal".

Trauma(5) is also cited as a predisposing cause to infection, and that of surgical operation is included. In the special case of tonsillectomy, the association between the raw area in the patient's throat and the occurrence of bulbar paralysis is definite, and indicates that under the special circumstances of such cases, the virus has access very readily to the bulb. Whether the trauma as such plays any part in this association is not clear. Russell(3) associates the injection of penicillin and vaccines with the location of paralysis.

Pregnancy has been noted as predisposing to infection(6).

Poliomyelitis occurs shortly after other infectious diseases in much the same way that infectious encephalitis occurs in association with these conditions(7).

Period/

### Period of Incubation.

The following paragraphs will demonstrate that there is difficulty in determining the end of the incubation period, by reason of variation in the clinical occurrences. There is also difficulty in deciding the date of apparent exposure. Horstmann and Paul(8) quote extremes of 3 and 35 days between apparent exposure and the onset of clinical symptoms. Casey(9) concluded that the average period of incubation was  $12.2 \pm 1.1$  days.

### Illness of Infection.

Transient pyrexia, often passing unnoticed, has been described after exposure to measles, mumps and smallpox. Naso-pharyngeal inflammation and pyrexia have been noted immediately following the presumed date of exposure to poliomyelitis. This is an uncommon occurrence, but one which makes it necessary to regard coryzal symptoms in contacts with suspicion. In such cases the virus has been demonstrated in the nasal secretions and faeces, and the victims must be regarded as precocious carriers.

A latent period, in which the patient is symptom free, follows this illness of infection.

### Invasion of the Nervous System.

Most cases, whether ultimately paralytic or not, present symptoms of diffuse involvement of the central nervous system as the first indication of the disease. For the purpose of description, this is often referred to as the meningeal phase.

During this phase, the most constant features are pyrexia, headache, rigidity of the spine and neck, and vomiting.

The pyrexia is moderate,  $101^{\circ}\text{F.}$  being a usual figure, and the duration seldom exceeds 48 to 96 hours. It may recur as the nervous manifestations mount in severity, giving a biphasic chart in which the zoologically minded have seen the likeness of the dromedary's humps.

Headache is almost always present in some degree, and may be severe. It has no characteristic distribution.

Nuchal and spinal rigidity appears to be due to the irritation of the central nervous system, and to differ from the muscular spasm so often found as a precursor to paralysis. Rigidity may be difficult to elicit with the patient only mildly ill and lying in bed. In a marked case, the raising of the head off the/



the pillow will provoke pain and resentment, but the degree of nuchal stiffness is usually less than that found in cases of septic or tuberculous meningitis. In a mild case it may be easy to flex the chin onto the chest, and Kernig's sign may be equivocal. Spinal rigidity is best elicited by asking the patient to sit up and put his legs over the side of the bed. Most patients suffering from pyrexia and headache will bend forward and support the head in the hands. Those with spinal rigidity will sit very erect, and will support the trunk by putting the hands in the middle of the bed far behind them. This sign is associated with the name of Amoss(10). If the patient is asked to kiss his knee, he cannot bend down to do so.

Vomiting is frequent, and appears to be due to central irritation.

The foregoing list is not exhaustive, but includes the signs and symptoms commonly found. When polio-myelitis is prevalent, the occurrence of headache, vomiting and pyrexia, makes it necessary for the physician to exclude this disease.

Other signs associated with the meningeal phase may be briefly recorded as follows:-

### 1. Respiratory.

There may be coryza, respiratory catarrh or sore throat.

### 2. Alimentary.

Constipation is rather more usual than diarrhoea.

### 3. Nervous.

The patient may be drowsy, restless and is characteristically irritable. Photophobia is common.

Particularly in cases which go on to paralysis, pain, hyperaesthesia and muscle spasm occur. Vesical paralysis, with retention of urine and overflow incontinence may be a very early occurrence.

The reflexes, deep and superficial, are not characteristic at this stage. As a rule the tendon reflexes are brisk and equal, inequality is suggestive, and absence may indicate the possible site of paralysis to come. Of the superficial reflexes, the plantar is usually flexor, while the abdominal reflexes may be depressed or absent. Nystagmus may be detected.

### Paralysis.

Although the appearance of flaccid paralysis in a voluntary muscle indicates the onset of this phase, certain/

certain of the nervous manifestations already mentioned strongly suggest that its onset is imminent. Muscular spasm is particularly suggestive.

The onset of paralysis usually occurs within about three days of the commencement of the illness. There is a tendency for it to occur relatively earlier in older children and adults, often within the first twenty-four hours. Early paralysis is not, however, confined to the older patient, as the name "morning paralysis"(11) used last century for the infant who went to bed well and woke up paralysed next morning indicates. In any outbreak, cases will occur in which paralysis is the first sign of illness. It is usual for paralysis to make its appearance before the temperature returns to normal. Occasionally its appearance is delayed for some days, but there is no symptom-free interval in such cases.

The location of the central lesion, with its consequent localisation of the paralysis, has been used as a means to accurate description. Two main divisions are recognised, the spinal form and the brain-stem form.

#### Spinal Poliomyelitis.

Localisation of the disease in the spinal cord gives rise to flaccid paralysis of voluntary muscles in the trunk and limbs. The lumbar enlargement, the cervical enlargement, and the intervening thoracic cord are involved in that order of frequency, though in many cases there is simultaneous involvement of more than one part of the cord. As a rule, paralysis is maximal at once, and tends to improve within a few days. In a few cases it may disappear entirely, but in the majority of cases there will be only partial improvement. At the other end of the scale, there are the cases in which no improvement occurs.

The severity of involvement may be assessed by electrical tests. Any muscle which responds to faradic stimulation three weeks after the onset of paralysis will recover completely. On the other hand, atrophy is rapid in those muscles whose motor nerves are destroyed, and the reaction of degeneration is found.

Paralysed muscles tend to undergo contracture, thereby causing deformities of trunk or limbs. Poliomyelitis, which has given rise to paralysis of muscles of the trunk, is thus a quite common cause of curvature of the spine in children. In the limbs, contracture may produce deformities, but where a whole limb is paralysed a complete flail is the more likely result. Vasomotor paralysis of a limb with subsequent retardation of growth is a recognised possibility.

Where paralysis is complete, the tendon reflexes associated with the paralysed muscles are absent. If a reflex does not disappear, or reappears early, recovery of the muscle will take place.

The cord lesion is not necessarily limited to the grey matter of the anterior horns, and there may be interference with afferent impulses in neighbouring columns, resulting in a varying degree of sensory disturbance.

In the previous paragraphs, the sequence of events has been described in which paralysis is maximal at once, and tends towards recovery, without recurrence. In a small number of cases the paralysis tends to ascend the cord, or to descend, the former being rather less uncommon. Cases of this type undergo paralysis followed by improvement serially in several sites. There is a considerable risk of ultimate fatal respiratory involvement.

#### Poliomyelitis Involving the Brain Stem.

The grey matter of the brain stem may be involved from the medulla upwards to the neighbourhood of the Red Nucleus. Lesions of the lower part give rise to bulbar paralysis, and those of the upper part to ocular and facial palsies.

Bulbar paralysis is by far the most serious of these. If the medulla is extensively involved, death rapidly ensues owing to damage to the vital centres. Failure of respiration is the most usual cause of death, but even if it is successfully controlled by the use of a respirator, death from circulatory failure usually occurs. Paralysis of the pharynx prevents the swallowing of saliva and other secretions, and may thus lead to an aspiration pneumonia. Laryngeal paralysis with loss of the power of phonation may occur, and so may interference with the mechanism of speaking.

Involvement of the upper part of the brain stem is of the same order of severity as involvement of the spinal cord. Death is unusual, and the extent of the residual paralysis depends on the severity of the lesions in the grey matter. The common residual defects are facial paralysis, spastic tremors, and ocular palsies with nystagmus.

#### Poliomyelitis of Other Types.

Various other types of poliomyelitis have been described. It should, however, be borne in mind that experimental poliomyelitis is limited to the spinal and brain stem types. This fact, coupled with increasing clinical experience of the disease, accounts for the growing tendency to regard some of these types as aetiologically distinct.



### 1. Cerebral Types.

Signs of encephalitis are followed by spastic paralysis of voluntary muscle. This condition seems to have much in common with post-vaccinal encephalitis, in fact it is not impossible that the occurrence of spastic paralysis in one limb along with flaccid paralysis in another, results from this condition following true poliomyelitis in the same way that it follows other infectious fevers. It is also possible that the condition is related to one of the other infectious encephalitides, such as lethargica, or to benign lymphocytic choriomeningitis. Van Rooyen and Rhodes consider that true polioencephalitis exists. Two recent cases of fatal polioencephalitis have been reported, thereby demonstrating the occurrence of the condition(12)(13).

### 2. Neuritic Types.

Here the presenting feature is muscular pain in one or other limb, with depression of tendon reflexes, but no paralysis. Either, it would seem, the condition is aetiologically distinct from poliomyelitis, or it represents the arrest of the disease on the verge of paralysis, as these pains are apt to indicate the site of paralysis before it appears.

### 3. Ataxic Type.

The presenting feature in this type is vertigo. It is accompanied by all the other early signs, but particularly vomiting, difficulty in standing and walking, partly due to weakness, and partly to giddiness. Nystagmus in some degree is to be expected, and there may be a degree of dysarthria. This type has been called the cerebellar type, but it could result from lesions in the Vestibular Nucleus, which would make it a variant of the brain stem form. Its occurrence in groups of cases and the bearing of this observation on its aetiology will be discussed when the 1947-48 outbreak is considered.

### Course of the Disease.

In paralytic cases, as has already been pointed out, the normal course is for the paralysis to be maximal shortly after its first appearance. The majority of cases show a substantial and rapid improvement, and in the course of a few weeks all traces of muscular impairment may have disappeared. If the initial paralysis is widespread or severe, such a degree of recovery is not to be expected, and some residual weakness follows.

The degree of recovery varies. In a few fortunate/



fortunate cases recovery may be substantial after extensive paralysis. On the whole, residual weakness is to be expected. In a fairly small number of cases there is no improvement, and the degree of paralysis remains unchanged. In a very limited number of cases there is extension of paralysis after its first appearance. As a rule, such cases are characterised by spread up the cord - the so-called Landry type. The progress may be arrested at any level, but often continues until respiration is embarrassed to such an extent that life cannot continue. Very occasionally the spread is in the opposite direction.

If there is residual paralysis, the case should pass to the orthopaedic surgeon as soon as the acute symptoms have died down, and the patient's progress thereafter becomes a problem in orthopaedics.

CLINICAL FEATURES REFERENCES.

- (1) Draper, G. (a) "Acute Poliomyelitis"  
Blakiston's, Sons & Co.  
Philadelphia, 1917.  
(b) Amer: J: Med: Sci: 184: 111,  
1932.  
(c) "Infantile Paralysis"  
D. Appleton Century Co:  
New York & London, 1935.
- (2) McCormick, W.J. (a) Canadian Med: Ass: J: 38: 260,  
1938.  
(b) Med: Record N.Y. 155: 89,  
1942.  
(c) Med: Record N.Y. 157: 414,  
1944.
- Heaslip, W.G. Australian J: Exper: Biol:  
Med: Sci: 16: 287, 1938.
- (3) Russell, W.R. (a) B.M.J. 2: 1023, 1947.  
(b) B.M.J. 1: 465, 1949.
- (4) Hargreaves, E.R. B.M.J. 2: 1021, 1948.
- (5) Lovett, R.W. & J.A.M.A. 51: 1677, 1908,  
Lucas, W.P. and many others.
- (6) McGoogan, L.S. Amer: J: Obst: & Gynec:  
24: 215, 1932.
- (7) Seeligmüller, A. Jahrb: f: Kinderk:  
12: 321, 1878; 13: 226 & 315,  
1879, and many others.
- (8) Horstmann, D.M. J.A.M.A. 135: 1947.  
& Paul, J.R.
- (9) Casey, A.E. J.A.M.A. 120: 805, 1942.
- (10) The name of Amoss is associated with numerous  
articles on the virology of poliomyelitis, not  
clinical features. His name is attached to the  
sign in text-books, e.g. Harries & Mittman, 3rd  
Edition, p.377.
- (11) West, C. London Med: Gaz: 32: 829,  
1843.
- (12) Jennings, G.H., B.M.J. 3: 210, 1949.  
Hamilton-Paterson,  
M.L. & MacCallum, F.O.
- (13) Howlett Kelleher, B.M.J. 3: 213, 1949.  
W., Bratton, A.B.  
& MacCallum, F.O.

## DIAGNOSIS.

The justification for making a diagnosis of poliomyelitis varies with the nature of the case, and the epidemic picture of the area. Consideration will therefore be given to paralytic and non-paralytic cases occurring in epidemic and non-epidemic areas.

### Diagnosis in Paralytic Cases.

Any case of flaccid paralysis of voluntary muscle, in child or adult, of sudden onset, with no obvious precursory symptoms and no obvious precipitating cause, requires a presumptive diagnosis of acute anterior poliomyelitis. This requirement is irrespective of other cases in the area, or of contact on the part of the victim with known cases or contacts, or visits to infected environments. That is to say, there should be no difficulty in recognising a paralytic case at any time.

### Diagnosis in Cases with no Paralysis.

It is in the case which occurs without paralysis, or before the onset of paralysis in which difficulty is likely. Here the epidemic history of the locality, and the patient's relationship to it, become important. In an area in which the disease is prevalent, the occurrence of several of the well known signs and symptoms together warrants a diagnosis of suspected poliomyelitis. The clinical findings on which weight can be placed are pyrexia, head-ache, vomiting, spinal rigidity, nuchal rigidity, altered reflexes, and perhaps angina. Jensen's figures for Denmark in 1934 were as follows(1):-

Pyrexia. . . . .	88.8%
Spinal rigidity . . . . .	85.4%
Nuchal rigidity . . . . .	34.9%
Angina . . . . .	56.4%
Reflex disturbance. . . . .	33.4%
Vomiting . . . . .	9.0%

In an area in which the disease is not at the time prevalent, diagnosis in the preparalytic stage is a matter of great difficulty for the practitioner. His accuracy may well depend on the thoroughness with which he examines his patient, and his experience in assessing his findings.

Both in epidemic periods, and at times of no special prevalence, borderline cases occur. In periods of prevalence, it seems not unreasonable to presume that illness, not incompatible with a diagnosis of abortive poliomyelitis, not clearly due to some other cause, occurring in a close contact, is in fact/

fact abortive poliomyelitis. When the disease is not prevalent, cases of meningism may be encountered with little to account for its appearance. This is perhaps more commonly found where there is also gastro-enteritis, than accompanying any other condition. Hitherto the occurrence of meningism has been considered as secondary to some other condition. An alternative that must be carefully considered is that both conditions result from the nervous irritation due to the virus of poliomyelitis.

It is generally recognised that the occurrence of a paralytic case in an area rapidly leads to the reporting of definite non-paralytic cases, the paralysis in the one being the evidence necessary to confirm the diagnosis in the others. This state of affairs will continue until a rapid and reliable confirmatory test for poliomyelitis becomes available, and only then will it become possible to estimate the incidence of the disease in the community.

### Differential Diagnosis.

The writers of medical text-books for the most part take the view that a certain diagnosis cannot be made in the preparalytic phase of the disease, and that the differential diagnosis involves distinction from other paralytic conditions. There is some truth in this contention, but I am of the opinion that an earlier diagnosis may be justified, and that early differentiation from some conditions can, indeed must, be made. The matter is therefore discussed under the following headings.

#### 1. Meningeal Conditions.

These include meningitis, both tuberculous and septic, and choriomeningitis.

Now that streptomycin is available, one of the most disastrous diagnostic errors is to confuse early tuberculous meningitis and non-paralytic poliomyelitis. To safeguard against such an occurrence, every suspected case should be admitted to hospital at once, and the cerebrospinal fluid examined. If the fluid is normal, the case can hardly be one of tuberculous meningitis. On the other hand, the diagnosis may be immediately made by the recovery of tubercle bacilli. It is the case with a moderate lymphocytosis, no organisms, and chemical constituents within normal limits that causes difficulty. Here it is best to repeat the examination after forty-eight hours. If the cells remain at the same level, and the protein is higher than usual, tuberculous meningitis must be suspected. The latest view is that the chloride content varies within too wide limits to be relied on, and that reduction/



reduction in the sugar is to be detected earlier and with greater constancy. Clinically, the following points are suggestive of tuberculous meningitis rather than of poliomyelitis. Nuchal rigidity. Positive Kernig's sign. Deterioration after temporary improvement following lumbar puncture.

The septic meningitic conditions should be readily distinguished by examination of the cerebrospinal fluid, with the recovery of the causal organism and the occurrence of granulocytosis.

Choriomeningitis is by some considered to be identical with non-paralytic poliomyelitis. A virus differing in character from that of poliomyelitis has been recovered by animal inoculation, and serum changes of a specific nature have been detected. The disease is therefore distinct. Its great clinical similarity has probably led to frequent diagnostic errors, especially where virological and serological methods have not been routine procedures.

## 2. Conditions Causing and Simulating Paralysis.

This group falls into two parts, conditions of nervous origin, and conditions originating outside the nervous system. The nervous conditions mentioned here are only those concerning which difficulty is likely to arise. Most other nervous conditions have characteristic features, such as insidious onset, sensory disturbance, or interference with sphincter control, which makes their further consideration unnecessary. The conditions to be considered are therefore encephalitis, polyneuritis, and acute brachial radiculitis.

### Encephalitis.

In the lethargic types, the onset is less acute, mental rather than meningeal symptoms are found, paralysis is uncommon, and the cerebrospinal fluid changes are inconstant. Where changes in the fluid do occur, they are similar to those found in poliomyelitis.

In the post-vaccinal type, with which are included those cases occurring after the exanthemata, after the initial and non-specific headache, vomiting and pyrexia, the patient becomes drowsy, and passes into coma. Convulsions are a likely occurrence, and various tremors are usual. In other respects there is little to guide the clinician. The most important single piece of information concerns the recent occurrence of one of the exanthemata or of vaccination, especially primary vaccination. The onset in post-vaccinal cases usually occurs about the tenth day.

### Polyneuritis/

### Polyneuritis.

The various toxic types do not need consideration. Landry's paralysis is very similar to infective polyneuritis, so much so that some observers consider the two conditions to be identical. In either case, there is a sudden onset, perhaps with general symptoms, sometimes accompanied by nerve pain and muscle tenderness, and followed by widespread, flaccid paralysis. This is usually progressive, and often ascending. Cases are often fatal owing to respiratory failure, but when recovery occurs it is complete. The cerebrospinal fluid protein is markedly raised but there is no pleocytosis.

### Acute Brachial Radiculitis.

This condition has achieved some prominence in the current decade. It occurs in the musculature of the shoulder girdle, in which painful paralysis occurs, recovering over a period of months. Sensory losses have also been reported. As the condition regresses on the one side, it may make its appearance on the other. The resemblance to poliomyelitis is not close, but as the condition is not very well-known, it might be a pitfall for the unwary.

The conditions originating outside the nervous system which might cause trouble to the diagnostician are characterised by the occurrence of acute pain, so that the part is voluntarily kept still to avoid causing pain. They include acute rheumatism, acute syphilitic epiphysitis, acute osteomyelitis, and scurvy. The list is not necessarily complete, but further elaboration is unnecessary. Adequate physical examination should prevent any error.

### 3. General Diseases.

Almost any acute illness has an onset which may imitate that of poliomyelitis, and to a large extent their differentiation is effected by observation for a few hours. In epidemic periods, there is always a tendency to label all cases poliomyelitis, and at other periods the other extreme is reached, and the case of so-called influenza later develops a limp. Much of this difficulty is more imaginary than real, and careful examination and consideration of all the circumstances will lead to the correct decision.

DIAGNOSIS REFERENCES.

(1) Jensen, C.

Poliomyelitis Epidemic in  
Denmark in 1934. Proc: Roy:  
Soc: Med: 28, 1007, 1935.



### TREATMENT.

If there is death of neurones, there can be no true recovery, and treatment must be directed towards minimising the occurrence of paralysis, if this be possible, and minimising its effects if it occurs. There is, in fact, no fundamental difference between the treatment meted out to Sir Walter Scott, and to my grandfather, and that available to-day. There have, of course, been refinements of detail. In the acute stage, the treatment is symptomatic, in the early paralytic stage, conservative, and in the late paralytic stage, when no further recovery can be expected, it is aimed at stabilisation and the avoidance of deformity.

There is at present no specific treatment. Fifteen years ago, convalescent serum had a considerable vogue, but later observations demonstrated that it was without effect unless given before the onset of symptoms. That is to say serum-prophylaxis is possible, but not serum-therapy.

The handling of this disease is therefore discussed under its various stages - the acute phase, early paralytic period, orthopaedics, respiratory failure, complications and special considerations.

#### Management in the Acute Phase.

The observations of Russell(1) and Hargreaves(2), already referred to, demonstrate the need for prompt and adequate action, if severe paralysis is to be avoided. Further, it should be stressed that, in epidemic periods, it is wise to err on the safe side, by instituting action suited to cases of poliomyelitis, rather than to wait for the diagnosis to become evident.

The patient should in nearly every case be hospitalised, as in very few homes is skilled day and night nursing available. The patient should be in bed, preferably in a single room, and in any case in quiet surroundings. The minimum of visits and examination are the rule.

Sleep must be secured, and for this purpose suitable doses of chloral and bromide are effective.

Pain, the fear of pain, and hyperaesthesia are common, and must be combatted. Aspirin in four-hourly doses is usually adequate medication, and with the application of heat locally, this condition is usually well controlled.

Attention to the bowels is on routine lines, but the/



the possibility of retention of urine, and resultant restlessness, must be kept in view.

#### Management of the Early Paralytic Case.

When paralysis makes its appearance, the patient should come under the supervision of an orthopaedic surgeon. The general principle to be followed is to secure immobilisation of the affected part in the optimum position until all signs of acute illness have passed.

If the paralysis is in the upper limb, immobilisation is the most important point, and positioning only of particular importance in deltoid paralysis. Here it is essential to keep the arm abducted.

If the paralysis is in the lower limb, the first point to secure is that the patient is placed flat on his back, and kept there, or there is no sound foundation for other measures of fixation. The optimum position for the lower limb is in adduction, extension but for a small pillow below the knee, and medial rotation. The foot must be kept in the normal position, and must not be allowed to flex into the dropped position.

When the musculature of the trunk is involved, the sequel to be avoided is spinal curvature. The patient must therefore be kept not only flat but straight.

#### Orthopaedic Treatment and Rehabilitation.

When the signs of acute illness have passed off, the first phase of orthopaedic remedial treatment commences. This is aimed at securing, by means of appropriate splints, plaster casts, or other appliances, the immobilisation of the affected part in such a way as to guard against deformity, and put the affected muscles in the best position for recovery. Physiotherapy is most important in this stage, and should include massage, movement, and the application of heat.

A patient whose paralysis is not extensive or severe may reach this stage while still in the isolation hospital, and can be discharged home at the end of his period of seclusion, if suitable measures to deal with the paralysed part have been taken. Where, however, the paralysis is severe or extensive, it is in the patient's interest to have him transferred to a fully equipped orthopaedic unit. Here the state of the muscles involved can be readily assessed by electrical methods, and in addition to the usual physiotherapeutic methods, a great deal of re-education can/

can be carried out. Another important point is that the education of children can receive attention, whereas the severely crippled child at home is liable to suffer in this respect.

It is generally considered that no further recovery takes place after the lapse of two years, and little after the lapse of six months. Undoubtedly this is the rule, but I have received information of recovery after much longer periods in a very limited number of cases(3). Many interesting problems concerning the regeneration of nerve tissue arise from these cases, and it will be further discussed in the section on prognosis.

It is not my purpose to enter into details of subsequent orthopaedic treatment. For the purpose of this study it will be sufficient to point out that, once it has become clear that there will be no further recovery, treatment is directed towards the remedy of deformity, the stabilisation of weak limbs, and the restoration as far as possible of lost movements. These aims are achieved by such operative procedures as tenotomy, tendon transplantation, or arthrodesis, accompanied if necessary by the application of supports. Except in the refinements of technique, there is no advance on the tenotomy and leg-irons which were the lot of my grandfather.

#### Management of Respiratory Failure.

As most cases of respiratory failure are consequent upon bulbar involvement, the other manifestations of this type of the disease will be considered here.

Whatever the cause of respiratory paralysis, the only effective treatment is continuous artificial respiration until the muscles recover, or, if they do not recover, for life. As continuous artificial respiration is only practicable with a mechanical device, the types of respirator in use at the present day will be outlined.

##### 1. Respirators Designed to Exert a Negative External Pressure.

This group includes the Both respirator, widely gifted to hospitals by Lord Nuffield. It consists of a box in size and shape closely resembling a coffin, in which the patient is placed, with the head protruding from the end, and an airtight junction at the neck. A large bellows is attached to the box by a rigid hose-pipe, and the air is alternately extracted from the box and allowed to flow in again.

## 2. Respirators Designed to Exert a Positive External Pressure.

This group includes the Bragg-Paul apparatus. In this type the thorax is encased in a rubber bag, into which the bellows pumps air under pressure allowing it to flow out again. The outfit is less cumbersome than those of the previous type.

## 3. Respirators Designed to Exert a Positive Internal Pressure.

In this type, the thoracic bag of the Bragg-Paul apparatus is replaced by a mask to cover the face. The air is forced into the lungs per vias naturales, and allowed to flow out again.

Respiratory failure due to bulbar paralysis is likely to be accompanied by cardiac failure, which must be dealt with symptomatically, and by paralysis of the palate, pharynx, and larynx. Attention must therefore be given to denying the buccal secretions access to the respiratory tract. To achieve this, the foot of the bed or respirator is raised, and the secretion removed by swabbing or suction apparatus. In addition, feeding by stomach tube, or per rectum will be necessary.

## Complications and Safeguards.

Apart from intercurrent illness, there is practically only one complication - respiratory infection.

In the event of any interference with the free movement of the chest, there is an immediate risk of broncho-pneumonia. Consequently it is essential to keep a close watch on the respiratory rate, and to commence artificial respiration at the first sign of respiratory paralysis.

The risk of aspiration pneumonia in case of pharyngeal paralysis has been noted.

There is some evidence that paralysed children, congregated together in an orthopaedic unit, are particularly liable to contact respiratory disorders, including the whole range of infectious diseases(4). This would appear to be remediable to a large extent by isolation of new admissions, diphtheria immunisation, avoidance of overcrowding in the wards, and the immediate removal to isolation of any patient showing signs of coryza.

The principal safeguards concern the avoidance of the spread of infection by secretions from the nose and/

and mouth, or in faeces and urine. Nurses in the fever hospital should wear masks when attending cases within seven days of the onset. All rags used as handkerchiefs must be burnt. Cutlery and crockery must be effectively sterilised. The precautions for the safe disposal of faeces and urine are those required in enteric fever. Excreta must be allowed to stand in contact with a suitable antiseptic before discharge. Bedpans and urinals require sterilisation. Soiled linen must be soaked in disinfectant. The nurse must wear a gown while attending the patient, and wash carefully after finishing with each patient. As the excreta may remain infective for three months, precautions concerning them are required in the orthopaedic unit as well as in the fever hospital.



TREATMENT REFERENCES.

- (1) Russell, W.R.      (a) B.M.J. 2:1947, P. 1023.  
                              (b)            1:1949, p. 465.
- (2) Hargreaves, E.R.      B.M.J. 2:1948, p. 1021.
- (3) Mr. Hay,              Personal communication:  
    Stracathro Hospital.
- (4)      Ditto.              Ditto.

## PROGNOSIS.

The prognosis in poliomyelitis depends on the stage and site of the disease. It is also concerned with danger to life, the likelihood of paralysis, and the chances of recovery from paralysis if this has set in.

### Danger to Life.

The quotation of a percentage mortality rate is of little significance, as it depends on the types of case included. If only cases of proved paralysis are taken into account, there is a death rate of from 5% to 25%. This wide range of variation is caused by the differing age groups affected, and differences in the anatomical types of disease. It is surprising how wide these variations may be from one epidemic to another.

#### 1. Age.

The most favoured age group is that from one to ten years. There is evidence that, by comparison, the chances for infants and adults are much less good than for children and adolescents..

#### 2. Spinal Poliomyelitis.

The risk in cases with paralysis of spinal origin is that spread to the cervical enlargement will lead to paralysis of the diaphragm and intercostal muscles. Untreated, this is naturally a highly fatal occurrence, but the prompt use of the respirator reduces this risk to small proportions. If the respiratory musculature is not involved, uncomplicated spinal poliomyelitis is not a fatal disease.

#### 3. Brainstem Poliomyelitis.

In this type the risk is almost confined to the bulbar type. Here the fatality rate is high, and death is liable to occur with tragic rapidity, perhaps within forty-eight hours of the onset. Of bulbar cases, three-quarters end fatally. Of uncomplicated brainstem paralysees, other than bulbar cases, the vast majority recover.

#### 4. Polioencephalitis.

The arguments against attributing any cases of encephalitis to the virus of poliomyelitis have been noted. I do not find myself wholly satisfied that the condition does not exist, and therefore give it brief notice here. Any encephalitic condition is serious, and carries a serious risk of death or crippling/

crippling involvement of the nervous system. I have found little concerning the prognosis of polioencephalitis in the literature. From a very limited personal experience I am disposed to consider the outlook gloomy, though the disease may be less fatal than other forms of encephalitis. I find references(1) to it as a cause of spastic palsies, and know of a case in which epilepsy has followed an illness diagnosed as polioencephalitis.

#### Danger of the Onset of Paralysis in the Non-paralytic Case.

Attention has already been drawn to the need for rest, from the moment that symptoms make their appearance, in order that paralysis may be avoided. It would appear to be a legitimate deduction, therefore, that where such rest has not been maintained, there is greater risk of the onset of paralysis, the recent papers of Russell(2) and Hargreaves(3) confirm this.

When there is marked illness without paralysis, the onset of paralysis becomes less likely as time passes. It usually makes itself apparent within three or four days of the onset of symptoms, but occasionally is delayed beyond this period.

The abatement of the initial symptoms without the appearance of paralysis is a very favourable sign. I have yet to see a case of paralysis unless its onset was linked by continuity of symptoms with the onset of disease. Where a true relapse takes place, I presume that there may be an interval, in which there are no apparent symptoms, between the initial illness and the relapse.

#### Outlook in Paralytic Cases.

Apart from the risk to life, the main question arising in the paralytic case is how much recovery is to be expected. With this is naturally associated the whole economic outlook for the victim.

The matter of immediate recovery has already been dealt with. Let it suffice here to say that most cases show substantial recovery, and that the expectation of recovery of a particular muscle is to be judged by its associated tendon reflex - if any - and its electrical reactions.

The ultimate prognosis depends on the location of the paralysis, its severity, and the skill and determination of those who undertake the patient's rehabilitation.

It is generally assumed that there can be no recovery/

recovery after the death of a neurone, and that any recovery of damaged but not dead neurones will take place within two years of the onset of disease. I have recently heard of a number of cases, none of which I have myself seen, in which recovery has either continued after the end of two years, and that to a substantial degree, or in which it has commenced at a much later date(4). These cases may be freaks of nature, in which case there is nothing to be learned from them. They may be cases in which there was extensive non-fatal damage to neurones. In its turn this gives rise to other possibilities. Firstly, the recovery of the neurones may have taken much longer than is commonly the case. Secondly, though the neurones had recovered, the patient suffered from a condition akin to hysteria, which prevented the use of the recovered neurones. Other possible explanations include the assumption that dead neurones can be replaced, or what is perhaps less unlikely, that under certain circumstances an axon can divide, the branch taking the path of the axon of the dead neurone. If the division of the axon took place in the course of the peripheral nerve this would not call for too great a stretch of the imagination. It is generally held that there is no regeneration of axons within the central nervous system. The matter is speculative, and the number of cases exceedingly small. I mention them solely to underline the need for determined perseverance on the part of doctor, patient and physio-therapist alike(5).



PROGNOSIS REFERENCES.

- (1) Strümpell, A. Allg: Wien: med: Ztg:  
29: 612, 1884.
- Marie, P. Progres med: 13: 167, 1885.
- (2) Russell, W.R. (a) B.M.J. 2:1947. p. 1023.  
(b) B.M.J. 1:1949. p. 465.
- (3) Hargreaves, E.R. B.M.J. 2:1948. p. 1021.
- (4) Hay, Stracathro Hospital, Personal communication.
- (5) Sugar & Gerard. J: Neurophys: 1: 7, 1938.  
J: Neurophys: 3: 1, 1940.
- Young, J.Z. Physiol: Reviews. 22: 318.
- Weddell, G. J: of Anat: 75: 346.
- Porter & Hart. Amer: J: Physiol: 66: 391.
- Denny-Brown,  
Eccles, Liddell  
& Sherrington. "Reflex Action in the Spinal  
Cord"

### PREVENTIVE MEASURES.

Mention has already been made of the need to isolate patients, and for the disinfection of their excreta and nasal secretions. These are matters of hospital routine, and need no repetition.

The most important measure for dealing with an outbreak is accurate information concerning it. This is secured by co-operation between the health authority and the doctors in the area. The former must keep the doctors posted concerning the likelihood of poliomyelitis, and its progress in the country. The latter must be on the alert to recognise the disease if confronted with it, and to report its occurrence at once. A good understanding, and mutual respect between the two parties is most essential. As far as the health authority is concerned, this co-operation is perhaps most readily secured by the provision of hospital services for the prompt admission and investigation of cases, for laboratory services, and for the provision of consultants who will take the responsibility for diagnosis in cases of difficulty.

The community prophylaxis falls into two parts - contacts, and food and drink.

#### Contacts.

In a town of any size, the isolation cannot go beyond the family, and often not as far as that. It is difficult to restrict the movements of the healthy adult. It would, however, be justifiable, and, by approach to the employer, easy, to keep a domiciliary contact from engaging in the food distributive trades.

In the country, it is probably easier to secure the isolation of a household, though this isolation may often be less effective than it is thought to be. Water and milk have to be carried from places where they are available to others, and the van is now an accepted part of country life in Great Britain.

In some parts of the world it is possible for the health authority to wield a power that would not be borne with in Great Britain. In Northern Rhodesia, the spread of poliomyelitis has been checked by putting a cordon round an infected area. This practice derives from the measures taken there and in neighbouring territories for the prevention of the spread of smallpox(1).

It is the unknown contact who may be dangerous as a carrier. For that reason all gatherings have to be considered carefully. The most contentious is the school - is it to remain open or be closed? Every Medical/

Medical Officer of Health must make up his mind on this subject in the light of local circumstances. If he is satisfied that school attendance constitutes a risk, then the school in question must be closed. It is, however, probably desirable to discourage unregulated gatherings of children, that is, gatherings not under the immediate control of the health services of the locality. Children should be kept away from picture houses, Sunday schools, and youth organisations of all kinds. Strenuous games should also be discouraged. On this ground and also on the ground that they conduce to the spread of naso-pharyngeal infections, swimming-baths should be closed to children.

The problem in the residential school is of a different nature. Here, there is a closed community, the members of which cannot be extensively isolated from each other. If poliomyelitis appears within the community, closure is necessary, the pupils being sent home in isolation, to continue isolation at home for three weeks. If the source of infection is outside the community, it is justifiable to await developments.

#### Foodstuffs and Drink.

I have already suggested that a community with a high standard of hygiene appears to be particularly liable to the ravages of poliomyelitis, and that this high standard tends to interfere with natural immunisation by exposure. While this may be true, it does not imply a disbelief in hygienic methods, least of all when there is an outbreak of poliomyelitis.

At all times, the basis of community hygiene must be the sanitary disposal of excrement, the provision of a safe water supply, and the cleanly handling of food and drink.

At this date, a high standard of sewage disposal is usual in public undertakings, though in rural communities the earth closet is still a common feature. Similarly, water supplies are, on the whole, satisfactory in quality. There is therefore little to do with them beyond keeping a watchful eye on the outbreak, to see if the distribution of cases incriminates either.

With regard to food supplies, it is unlikely that greater attention to the hygiene of shops, other than to eradicate flies, could have any effect. Again, the proper course is to keep a close watch on the distribution of the outbreak, with a view to detecting the presence of a carrier who contaminates by handling.

Evidence has already been mentioned of milk as a vehicle of spread. Some authorities go as far as to demand the pasteurisation of all milk supplies during an/

an outbreak. A more moderate view which commends itself to me to be justified, namely watching for any evidence that milk is the vehicle, and then taking action.

#### Immunisation.

No worthwhile procedure has so far been evolved. Attempts have been made with killed and attenuated vaccines. The former are valueless, and the latter so far too dangerous. Undoubtedly the answer to the problem lies in this direction. No therapy can reasonably be expected to banish poliomyelitis as a paralytic, and fatal disease. Immunisation is the only procedure which can reasonably be expected to prevent mankind from the dread of repeated epidemics.



PREVENTIVE MEASURES REFERENCES.

(1) Gear, J.H.S. &  
Rodger, M.L.

Manuscript quoted by  
Rhodes, A.J. Bull: Hyg:  
22: 6, 1947.

## THE OUTBREAK OF POLIOMYELITIS IN MORAY AND NAIRN.

### 1947 AND AFTER.

The poliomyelitis outbreak which commenced in Great Britain in 1947, and of which that in Moray and Nairn formed a small part, cannot be treated as an isolated incident. It is therefore necessary to review briefly certain aspects of the preceding years.

From September, 1939, when the first bomb fell on Warsaw, till the Japanese capitulation in 1945, much of the inhabited land surface of the globe was desolated by armed combat. In no part of the world was the struggle unfelt. The consequences of this upheaval, apart from the loss of life and property, have been the lowering of standards of living, and the intermingling of populations on an unprecedented scale.

Apart from the havoc wrought by enemy air attacks, the island of Great Britain suffered relatively little. There were overcrowding and evacuation. There were food shortages. The flower of our manhood went overseas in the Navy, Army and Air Force. We welcomed to our ranks refugees from Europe, and armed forces from the Colonies, Dominions, and the New World. During these years, and for some time after, there were in this country prisoners of war from the Axis countries.

This is a formidable list of unusual events, if taken from its context. But in relation to the scenes of carnage and wreckage in other parts of the globe it presents a humdrum air of near-normalcy. Support for the latter view-point is found in the satisfactory vital statistics of the country. The death-rates of women in childbirth, and of infants in the first year of life, continued to fall. There were signs of strain it is true. The incidence of tuberculosis increased. But there was no epidemic of a major nature other than that of scabies, and this condition, though having a high nuisance value, cannot be numbered amongst the dangerous diseases.

By 1947, most of our guests - welcome and unwelcome, voluntary or involuntary - had returned whence they came. Great Britain was again inhabited mainly by Britons for the first time for nearly ten years. And in this year the country encounters its first great epidemic of poliomyelitis. Why?

It is attractive to associate this happening with our visitors, with our shortages of food, with our weather, and even perhaps with the alleged misdeeds of the Government of the day. In the following pages an attempt will be made to answer the question in the light of intimate experience of a small section of the problem.

DISTRIBUTION OF POLIOMYELITIS IN THE WORLD,  
1939 TO 1946.

Rhodes' very excellent summary of the principal outbreaks in the world makes the task of presenting a survey of world distribution an easy one.

In the United States of America, outbreaks are particularly well documented, presumably because they have been particularly numerous. As a result, it is known that outbreaks of some magnitude occurred in 1939 in Detroit, South Carolina and in Tennessee; in 1940 in Washington; in 1941 in Alabama, Mississippi and Ohio; in 1942 in New York State; in 1943 in Chicago, Kansas, Los Angeles, Texas and Utah; in 1944 there was a recurrence in New York State, and fresh outbreaks in Buffalo, North Carolina and Virginia; in 1945 there was a recurrence in Utah, and a new outbreak in New Jersey as well as in some service establishments.

During the same period there were outbreaks in Canada. Manitoba was involved in 1941, and its capital, Winnipeg, in the following year.

The countries of Central and South America and the Islands of the West Indies have been endemic centres for the whole of the period, and few, if any of them have been entirely free of epidemics or increases in prevalence in that period.

In European countries, the period was one of substantial prevalence. Denmark was involved in 1942 and 1944; Finland in 1940, 1944 and 1945; in Sweden the experience was similar with outbreaks in 1943, 1944 and 1945. Iceland was affected in 1945. France was involved in outbreaks in 1943 and 1945. Germany sustained outbreaks in 1939, 1941 and 1942, and Berlin sustained a serious outbreak in 1947, after that in Great Britain, and possibly associated with it. Belgium and Holland were visited in 1939, 1943 and 1945. Switzerland was affected in 1941, 1942 and 1944. In fact, throughout Western Europe there was a series of local outbreaks, and a steadily high incidence during the period of the war.

In the Near and Middle East, the area from Malta to India, the customary incidences prevailed amongst the local populations, in which the disease was clearly endemic. These were punctuated by outbreaks amongst service personnel, British, Dominion and American alike. The outbreak in Malta in 1942 to 1943 involved the local populace as well as the garrison. The incidence amongst service personnel, no matter what their countries of origin, was roughly ten times as great as that in formations in those home countries.

In Africa, there was the usual endemic prevalence throughout the continent, with a sharp outbreak in the Union of South Africa, where some 1300 cases occurred between February 1944 and June 1945. The suggestion was made at the time that returning service personnel had introduced a new strain of virus.

In point of time the outbreak in St. Helena was simultaneous with that in the Union of South Africa, and, in view of the normal sea-borne traffic between the two, probably a part of the same occurrence. The same may be said of the outbreak in Mauritius in 1945.

Australasia and New Zealand were relatively free from outbreaks during this period. The exceptions are that in New South Wales in 1945 and 1946, and in Singapore at the same time. If it be conceded that the outbreak in South Africa, St. Helena, and Mauritius resulted from the introduction of a virus from the area where Europe, Asia and Africa meet, the same possibility concerning the Far East must be seriously entertained.

The experience of Great Britain in 1947 seems to bear interpretation on similar lines. During the decade 1930 to 1939, there were several minor outbreaks. But taken by and large, the incidence followed that of Continental Europe. With the Nazi conquest of Western Europe in 1940, and the erection of the Westwall fortifications, another barrier, and that an epidemiological one, was set up. There were small increases of prevalence in 1940 and again in 1945, but there was no counterpart to the experiences of the countries immediately across the narrow seas. From 1945, intercourse with Western Europe was steadily increased, and our forces in India and the Far East returned. The opportunity of importing virus was undoubtedly present, and our experience of isolation for half a decade could have lowered our national resistance.

(See Chart I).



## HISTORY OF POLIOMYELITIS IN GREAT BRITAIN.

The early history of poliomyelitis in Great Britain has already been outlined in the introductory summary. The steady occurrence of sporadic cases is indicated, along with the occasional occurrence of an outbreak of local prevalence. This state of affairs persisted throughout the nineteenth century.

Interest in the condition was, however, awakening, and reports of cases and outbreaks appeared with increasing frequency in the medical press. Between the years 1897 and 1910, McNalty mentions outbreaks in Bristol, Cumberland, Dorset, Essex, Gloucestershire, Herefordshire, Leicestershire, London, Northumberland, Nottinghamshire, and Yorkshire. None of these appears to have involved more than a handful of cases(1).

In connection with the year 1911, McNalty, in the same paper, uses the word "epidemic" for the first time. Reports of substantial prevalence came from many countries in England and Wales, from Cornwall to Westmoreland, and from Wales to Suffolk. The condition was not at that time notifiable, but the experience of that year caused the Board of Health in England to make notification compulsory in 1912, with 1913 the first complete calendar year for which records are available. The notifications for the next twenty years indicate a prevalence of 641 notified cases per annum. The extremes were 228 in 1918 and 1297 in 1926, a year generally credited with epidemic prevalence. It is interesting to note that the notifications for the years 1913 to 1918, inclusive, numbered 2800, while those for 1928 to 1932, the years corresponding to 1926 as the former quinquennium corresponds to 1911, numbered 2806. It seems reasonable to assume, therefore, that the magnitude of the epidemic of 1911 corresponded closely with that of 1926 when 1297 cases occurred.

Up to this time there is virtually no mention of the occurrence of the condition in Scotland - apart from Sir Walter Scott and my relatives. The epidemic of 1911 does not seem to have reached the northern kingdom in epidemic form, and the Scottish Board of Health did not feel the need for notification for some years. When notification came in 1918, it was a permissive measure, of which hardly any of the Local Authorities availed themselves. Only in 1926 was notification made obligatory in Scotland, and not for another dozen years was there a substantial prevalence.

The views of the Edinburgh Medical School, as expressed in lectures to students up till the year 1930 or thereby are comprehensible, though now clearly based on false premises. The teaching was that no outbreak in Scotland ever affected more than three persons/

persons. If, therefore, three cases of paralysis occurred, the outbreak was over. When pressed to account for this, the explanation offered was that Scottish immunity was at a higher level, the underlying immunological difference being unknown. In the light of subsequent events, the reason appears to have been that the virus had not at that time deployed itself for a full-scale invasion of Scotland.

One of the first reported outbreaks in Scotland occurred in 1928. Halliday(2) described a group of cases occurring in a circumscribed locality in the City of Glasgow, and pointed out that a number of other similar groups had occurred. The total notifications in the city in the year 1928 were 112.

The inference to be drawn from the foregoing experience, differing as it does between the two kingdoms, appears to be as follows:-

England and Wales became affected by the virus of poliomyelitis in the first decade of this century. From the commencement of the second decade to middle of the fifth, a moderate degree of prevalence was experienced, showing itself in minor epidemics on a national scale in at least two years.

Scotland was not involved along with England and Wales at the outset and prevalence on a similar scale was delayed by at least thirty years. When, therefore, the two countries were simultaneously and equally exposed in 1947, the herd immunity in the northern kingdom proved to be substantially less than that in the southern, and a substantially higher rate of notification was consequently experienced.

#### Relationship to Weather Conditions.

It may be that the occurrence of the first serious epidemic in the summer of 1911, now almost legendary for its magnificent weather, has caused observers in this country to pay undue attention to meteorological factors. It is worth giving the facts a brief scrutiny. The summer of 1910 was lamentable, with rain and floods. There was no exceptionally severe weather in the winter 1910 to 1911. The summer of 1911 was dry and hot, with water shortages. Ten years later, in 1920, there was a lamentable summer, with rain and floods, the subsequent winter was of normal severity, and the summer of 1921 was again magnificently fine, with serious drought. There was, however, no epidemic. The summer of 1925 was good, with a period of fine weather in the early part. The winter of 1925 to 1926 was fairly severe at times, and part of Loch Lomond was frozen for the first time for many years. The summer of 1926 was graced with mixed weather. An Australian cricket team was touring this country/

country. Two of the Test Matches were abandoned after little play on account of rain, and rain greatly affected the result of the final match. This year also saw the greatest recorded prevalence to date in the southern kingdom. The next year of outstanding meteorological interest was 1933. The prolonged fine weather rivalled that of 1921 or 1911. In the previous year there were fine spells, punctuated by rain, but on balance the fine weather prevailed. The intervening winter was not outstanding. Notifications of poliomyelitis in England and Wales were slightly above the average, but nothing of the nature of an epidemic occurred. The year 1938 was one of general prevalence of poliomyelitis on both sides of the Border. The summer of 1937 was perhaps above the average, but not outstandingly so, the winter of 1937 to 1938 was unusually mild, and the spring early. The summer months were dry but not unduly warm. From this time on there were four years with hot dry summers and cold dry winters. These were the years of Hitler's war on Poland, on the Low Countries, on France, of the Battle of Britain and the blitz. On this side of the Channel there was no undue prevalence of poliomyelitis. From 1943 to 1945, the weather returned to more average behaviour, with the admixture of wet and fine, rain and frost, each in their appropriate season, to which we are accustomed. In 1946, there was a return to summer conditions which almost deserve the term lamentable, but these wet conditions did not involve the whole country equally. The year 1947 was ushered in with a period of cold weather. In the north, the cold was severe, in the south it was of unparalleled severity. In the north, spring was late and reluctant, and until mid-summer the cold threatened to return. From mid-July, there was a period of unparalleled heat and dryness, not relieved for six weeks, and repeated in a lesser degree in September and October. In the south, the warm weather set in earlier than in the north, and continued with little or no break into the autumn. Between the severity of winter and the torrid summer there was an interregnum of severe floods. There was, in fact over the whole country, and over England in particular, an approach for nearly twelve months on end to the climatic conditions normally prevailing in the northern states of America, or across the North Sea in Scandinavia. In this period there occurred an epidemic involving six times as many persons as were attacked in any previous outbreak.

Gard(3) relates the seasonal incidence in Sweden to the temperature and to the humidity as indicated by rainfall. The occurrence of frosty weather in the autumn brings the year's prevalence to a close, and the cessation of this type of weather in spring re-opens the season's activities. It should be remembered that the autumnal decline, though less marked/



marked, occurs in areas where frost is a transient rarity.

Gard also relates the periods of local prevalence which go to make up the national picture, to the rainfall. The peak of prevalence occurs, he states, two to three weeks after the rainfall has been excessive. Rhodes(4) suggests meteorological investigations, and that the relationship between humidity and droplet infection should be studied. It would be of interest to find confirmatory reports of Gard's observations from North America. The vagaries of our British climate would make a relationship between excessive rainfall and the incidence of poliomyelitis very difficult to detect. Now that this country has been as it were "indoctrinated" with the virus of poliomyelitis, the presence of the virus can be assumed in all parts of the country for some years to come, whereas there must always be doubt as to its presence in strength in previous years, say 1921, or 1933.

A justifiable conclusion concerning meteorological factors appears to be that dryness favours the onset of prevalence, and that previous rainy weather or frosty weather may favour the onset. I for one shall continue to believe that the combination of rain, frost and dry heat, in that order in 1946 and 1947, materially assisted the pandemic of the latter year, until an outbreak of similar severity occurs under totally different weather conditions.



## HISTORY OF POLIOMYELITIS IN GREAT BRITAIN

### REFERENCES.

- (1) McNalty, A.S. B.M.J. Vol. 2: 1936, p.57.
- (2) Halliday, J.L. Trans: Med-Chir: Soc: Glasgow.  
25: 56, 1930-31.
- (3) Gard, S. Bull: Office Internat: d'Hyg:  
Publique. V29, 269.
- Bull: Office Internat: d'Hyg:  
Publique. V30, 933.
- (4) Rhodes, A.J. Bull: Hyg: V22: 6, 1947.

OUTBREAK OF 1947.Onset.

In England and Wales, the weekly notifications of poliomyelitis numbered less than 20 for the first 21 weeks of the year. Thereafter there was a steady rise in the notifications.

Although the total notifications remained steadily at a fairly low level during these weeks, local prevalence in several areas was discernible. For ease of demonstration the county boroughs have been included in their respective counties, and references to populations, cases or rates of incidence refer to the combined areas.

In six of these counties ten or more cases occurred in the twenty-week period under review, namely -

London County	26
Yorkshire, West Riding	19
Warwickshire	18
Middlesex	14
Lancashire	13
Essex	11

These counties lie in three main groups, the London Group comprising London County, Middlesex, and Essex, the Midland Group in which the only county unit was Warwickshire, and the Northern Group, consisting of Lancashire and the West Riding of Yorkshire.

At the same time as these local degrees of prevalence were occurring, no cases whatever were recorded in 13 English and 8 Welsh Counties. These Counties were in two main groups, the Eastern Midlands and rural Wales. Two smaller groups were noted in the rural north west, and in the upper area of the Upper Thames Valley.

To summarise, therefore, cases of poliomyelitis were already appearing in significant numbers in the main centres of urban population and industry, in London and the Home Counties, the Black Country, and the industrial North, while it was absent in those areas where towns are smaller and agriculture is more predominant. The virus was well placed in the populous centres to attack victims ready to hand, and to spread thence throughout the country carried by those who used the excellent communications available.

Weather in Relation to Area of Onset.

The six county areas already mentioned comprised three in the south - London, Middlesex, and Essex, one in/

in the Midlands - Warwickshire, and two in the North - Lancashire and the West Riding of Yorkshire. It has already been noted that the weather in the early part of 1947 was severe in the north and of unparalleled severity in the south. Relatively, the further south the severer conditions became. It may be that this finds itself in the higher case rates in the south, thus:-

	<u>Population</u>	<u>Cases</u>	<u>Rate per 1,000,000</u>
London Group	7,135,900	51	7.14
London County			
Middlesex			
Essex			
Midland Group	1,727,870	18	10.3
Warwickshire			
Northern Group	8,261,780	32	3.8
Lancashire			
Yorkshire W.R.			

#### Rise of the Outbreak.

In the twenty-second week of 1947, notifications in England and Wales exceeded 20 for the first time. This may be taken as the first definite upward movement of the epidemic wave. The first crest was reached twelve weeks later, in the thirty-third week, when 691 cases were notified. Three weeks later still, the two intervening weeks having shown slight recessions, a second, and slightly higher crest was reached with 708 notifications.

By this time, the main areas of prevalence were clearly discernible, namely London and the Home Counties, Lancashire, Cheshire and Yorkshire, particularly the West Riding, Warwickshire and Staffordshire, and Tyne-side.

In the south-eastern group of counties, the peaks lay between the thirty-second and thirty-seventh weeks, with a general tendency to coincide with the second national peak.

In the northern group, including the Tyne-side cases, several of the county peaks occurred as early as the thirty-first week. Lancashire in particular had no peak week for notifications, the figures for the thirty-second to thirty-seventh weeks inclusive hardly varying. Generally, there was a tendency to coincide with the first national peak.

In the Midlands, the tendency to a high and steady level of notifications over several weeks, including both national peaks, was also noted.

Generally/

Generally speaking, therefore, the outbreak followed the pattern suggested in the previous section. The first onslaught was experienced in those areas which had exhibited a prevalence in the earlier weeks of the year. These areas likewise experienced the greatest total weight of attack. From these areas the epidemic spread rapidly to contiguous and remote areas alike, choosing each fresh point of attack in a somewhat unpredictable manner. The time relation between the outbreaks in London, Lancashire and Glasgow is shown on Chart VI in the Appendix.

#### Decline.

From the peak of 708 notifications in the thirty-sixth week, there was a steady and progressive fall until the weekly figure was less than 100 in the forty-ninth week. Thereafter there was fluctuation around the mean of 65 to 70 notifications per week.

All the counties noted for early or marked epidemic prevalence continued to show notifications on a fairly substantial level to the end of the year.

Counties in the East Midlands, such as Cambridge, Ely, Huntingdon, Peterborough, and Rutland, showed at no time more than a slight prevalence of sporadic cases.

A few counties in widely separated areas, showed peak notifications in or after the fortieth week of the year. These included Bedford, Cornwall, Derby, Norfolk and perhaps Wiltshire. In Cornwall and Norfolk in particular was this autumnal peak discernible, occurring as it did in the forty-sixth week.

In Wales also, apart from the industrial south, the sporadic prevalence tended towards an autumn peak.

The general picture towards the close of the year is that of a woodland fire, smouldering extensively in the most affected areas, and surrounded by fringes of flame. (See Charts II and V).

#### Scotland in Relation to England and Wales.

The population of Scotland, at 5,100,000, is comparable with those of Lancashire, 4,848,690, and London, 3,109,240. In the first twenty weeks of 1947 there were 18 notifications of poliomyelitis in Scotland. This prevalence is comparable with that in the English areas cited in the same period.

The notifications were distributed as follows:-

Glasgow/



Glasgow	3
Airdrie	2
Lanarkshire	1
Greenock	1
Paisley	1
Kilmarnock	1
Ayr	2
Falkirk	1
Perth	1
Edinburgh	1
West Lothian	1
Mid Lothian	1
Aberdeenshire	2
Total	<hr/> 18 <hr/>

As in England, therefore, the incidence was in the urban and industrial areas. Cases occurred in or near all the main towns, Dundee excepted. The rural areas of the Highlands and Borders were free.

The main epidemic rise did not clearly show itself until the twenty-ninth week, when notifications totalled 19. The relationship in time between the epidemic in Scotland and England as whole may be seen in Chart V in the Appendix, and between London, Lancashire and Glasgow in Chart VI to which attention has already been drawn. The latter demonstrates an interval of approximately one week between the commencement of the sharp epidemic rise in London and Lancashire, and the same interval between Lancashire and Glasgow.

#### Moray and Nairn in Relation to Scotland.

The administrative county of Moray and Nairn lies on the southern shore of the Moray Firth. There is a coastal strip, approximately ten miles wide - the "Laich of Moray". To the south of this coastal plain the hills begin to rise, at first in gentle folds, but with the higher tops, which lie further inland, peering over. From these higher hills descend three large rivers, the Nairn, Findhorn and Spey from West to East. The largest of these, the Spey, rises ninety miles to the south-west, and its valley provides a convenient line for road and rail communication to the south. A smaller stream, the Lossie, drains the hills close to the coast, and discharges into the sea midway between the Spey and Findhorn.

The population is concentrated in the coastal plain, where the main towns lie. The Registrar-General's estimate for the middle of 1947 totalled 54,774. Of this, 83% was located in the coastal strip, and the remaining 17% was distributed in smaller communities in the upland areas, considerable tracts of/

of which are virtually unpeopled.

The main trunk road from Aberdeen to Inverness passes through the coastal belt, traversing the towns of Elgin, Forres and Nairn on its westerly way. The main road from Inverness to the South is joined by a feeder from Forres, which unites with a road of rather less importance from Elgin and the eastern coastal area.

The railways follow the same general pattern, with an east to west mainline through Elgin, Forres and Nairn, joined by a branch of the mainline from the south at Forres, and with a subsidiary connection to Elgin by way of Strathspey.

The countryside depends on agriculture and fisheries for its prosperity, and in the summer months shares with other highland and northern coastal areas in popularity with holiday-makers from the more urban and industrial south.

In fact, the area described is essentially similar to numerous other rural and coastal counties in Scotland. There is no obvious factor in its situation, or the pursuits of its inhabitants to account for the abnormal prevalence of poliomyelitis in 1947. (See Charts IV and VII).

#### Previous Experience of Poliomyelitis in Moray and Nairn.

In attempting to gauge the experience of poliomyelitis of the community in Moray and Nairn, and to deduce therefrom the state of basic immunity, enquiries have been made in three main directions. Not unnaturally the first source of information consulted was the Register of Notifications of Infectious Diseases.

Secondly, the older medical practitioners in the area, and indeed in the north-east of Scotland, have been approached, and their recollections of cases or outbreaks in the area ascertained. Thirdly, an attempt has been made to estimate the numbers now resident in Moray and Nairn, who suffer from residual paralysis due to poliomyelitis.

#### 1. Previous Notifications.

Registers of Notifications of Infectious Diseases prior to the year 1930 are no longer extant. For the years from 1931 to 1946 inclusive, I prepared the following table of notifications for my Annual Report for the year 1947. Approximate figures for population have been added.

Table/

Table of Notifications of Poliomyelitis, 1930 - 1946.

Year	Moray (20,000)	Elgin (9,000)	Forres (4,400)	Burghead (1,200)	Grantown (1,500)	Lossiemouth (4,300)	Rothies (1,200)	Nairnshire (4,500)	Nairn (4,400)	Total (49,500)
1930	-	-	-	-	-	-	-	-	-	-
1931	-	-	-	-	-	-	-	-	-	-
1932	-	-	-	-	-	-	-	-	-	-
1933	-	-	-	-	-	-	1	-	2	3
1934	1	-	-	-	-	-	-	-	-	1
1935	1	-	-	-	-	-	-	-	-	1
1936	-	-	-	-	-	-	-	-	-	-
1937	-	-	-	-	-	-	-	-	-	-
1938	-	-	-	-	1	-	-	-	-	1
1939	-	-	-	-	-	-	-	-	-	-
1940	-	-	-	-	-	-	-	-	-	-
1941	-	-	-	-	-	-	-	-	-	-
1942	-	-	-	-	-	-	-	-	-	-
1943	-	-	-	-	-	-	-	-	-	-
1944	1	-	-	-	-	-	-	1	-	2
1945	-	-	-	-	-	-	-	-	-	-
1946	-	-	-	-	-	-	-	-	-	-
Total	3	-	-	-	1	-	1	1	2	8

From this table it will be readily seen that, as far as notifications are concerned, few cases appear to have occurred in the seventeen years preceding the outbreak of 1947.

## 2. Recollections of the Older Medical Practitioners.

I must here declare my indebtedness to my professional colleagues in Moray and Nairn for the whole-hearted way they have tried to help me in the enquiries which have preceded this study. In spite of the goodwill, however, the recollections of remote events, which were not recorded in writing at the time, puts as much strain on the memory of the medical man as on that of the layman. I can therefore only consider these recollections to be accurate within a year or two, and not necessarily to cover all cases which occurred. In fact it would appear highly unlikely that any but well-marked cases of paralysis would be remembered as cases of poliomyelitis.

The consensus of opinion is, however, that there was increased prevalence around the years 1916 to 1918, another period of prevalence in the middle of the decade 1920 to 1929, probably associated with the years 1925 and 1926, and a further period of prevalence ten years later. The last-named appears in the six notifications in the years 1933 to 1938. I am inclined to the view that rather more cases occurred in this last period than were notified. In respect of the prevalence in 1944, this may have been preceded by a slight prevalence of non-paralytic cases in 1942 and 1943. I know of one case diagnosed as benign lymphocytic meningitis which occurred in 1942. The findings in the cerebrospinal fluid were consistent with this diagnosis, and also with one of poliomyelitis. No serological examinations were made. One other case was admitted about this time to the fever hospital, with a diagnosis of encephalitis. The cerebrospinal fluid was non-pathological, and the patient made a satisfactory recovery. This also could have been a case of poliomyelitis.

## 3. Known Cases of Residual Paralysis.

In the course of years of service in a fairly small community, the Medical Officer of Health naturally gets to know of cripples in the area. I know of about six cases in Moray and Nairn almost all of whom have partial paralysis of one or other leg, clearly associated with poliomyelitis. Most of the persons in this group are now reaching middle age, and were paralysed between 1916 and 1926.

A further cross-bearing suggested itself in the records of the North-Eastern Cripples Welfare Association/



Association. This voluntary organisation made a survey of cripples in the north-east of Scotland in 1942 and arranged for orthopaedic clinics and after-care. The survey showed thirty-one persons in 1942 who were believed to suffer from paralysis following poliomyelitis. The Association's work has now merged in that of the North-Eastern Regional Hospital Board, and the records are not fully available. As far as they go they show that only one in four was ultimately accepted as due to poliomyelitis.

These two methods of approach suggest that there are perhaps a dozen old cases of paralysis due to poliomyelitis resident in the area. Present residence does not, of course, demonstrate locality of infection. On the other hand, the information otherwise gathered suggests that the experience of Moray and Nairn was not grossly different from that of other similar rural parts of Scotland. If that is accepted, then the likelihood of inward migration balances that of outward migration, and the number of paralytics in the area prior to 1947 was a fair index of the prevalence of the disease.

The information contained in the foregoing paragraphs may be summarised by saying that in three months in 1947 many more cases occurred than in the previous thirty years or more. Further, about a decade had passed since there had been any great prevalence. The conclusion to be drawn, therefore, is that the community of Moray and Nairn was virtually unsalted, as far as poliomyelitis was concerned, in the earlier months of the year 1947.

#### The Weather in Moray and Nairn, 1946 to 1947.

There is little to add to the previous remarks concerning weather. Moray and Nairn shared in the wet spell in the harvest and autumn of 1946. The rainfall was, however, proportionate. That is to say, it did not exceed that of the area in a greater degree than occurred elsewhere. As the area has normally a low rainfall, the excess of the autumn in question did not lead to serious interference with the harvest, or to flooding.

When the severe spell of wintry weather set in early in 1947, it did so on account of an easterly stream of very cold air. This caused heavy snowfalls over much of the country. In the Laich of Moray, the fall hardly exceeded eight inches, as the high hills to the east and south captured the precipitation. The frost was keen and continuous, doing considerable damage to shrubs which had no covering of snow. The temperature was not as low as it has been on three occasions in the last seven years. During the month of February there was a succession of days of brilliant sunshine/

sunshine at the time when London did not see the sun for three weeks. In fact, though the cold was severe, its only unusual feature was its prolongation.

The thaw took place in March, but for many weeks the temperature remained very low. In the north-east of Scotland the "Gab of May" is a well-known cold spell. In 1947 it was particularly well-marked, and practically continuous with the winter.

Suddenly, about the beginning of June there was a brief spell of warm fine weather, quickly followed by another month of cold though fair conditions. Then, about 20th July, 1947, there started a period of scorching heat and continuous dryness. Temperatures in the coastal area of Moray seldom rise above 80°F, as a sea breeze sets in. The Meteorological Officer at the Fleet Air Arm Station at Lossiemouth has told me that it lowers the temperature there by 10°F for every fifteen minutes it blows, and that it raises the humidity rapidly. There is no other meteorological station in the area which would not be affected by the same conditions. Under the circumstances, no specific meteorological data have been put forward. In spite of the cooling sea breeze, and the coastal humidity, no rain fell in the whole month of August, 1947. There was some cold and rainy weather in the first half of September, 1947, but there was a return to warm and sunny conditions in the second half of the month, and in October. Thereafter, the weather partook of the autumnal and wintry season without any extremes.

#### Time Relationships.

For the purposes of placing the outbreak in Moray and Nairn in its proper time relationship by comparison with the outbreaks in other parts of Scotland, I have prepared Chart VII which will be found after the Appendix. In this chart the notifications of poliomyelitis in the Cities of Glasgow and Edinburgh, the County of Ayr, including the Burghs of Ayr and Kilmarnock, and the County of Dumfries, including its County Town.

Mention has already been made of the sharp rise which started in London, Lancashire, and Glasgow in the twenty-seventh, twenty-eighth, and twenty-ninth weeks of the year 1947 respectively. In four weeks following, notifications in Glasgow reached a peak, followed by two more at intervals of three and two weeks respectively, after which notifications declined rapidly.

In Ayrshire, the same picture is presented, although the graph shows a plateau rather than a series of peaks. The rise and fall of notifications is, however, simultaneous with that in Glasgow.

In Edinburgh, the rise in notifications did not commence till about the thirty-third week, and a single sharp summit was reached in the thirty-sixth week, followed by an equally sharp fall which synchronised with those in Glasgow and Ayrshire.

In Dumfries-shire, there was a slow rise to a plateau, coinciding with the peak in Edinburgh, followed by a sharp peak in the fortieth week, when the previously mentioned areas were showing substantial declines. It is true that at this time there was a slight check in the decline in Glasgow, but only to the extent of two cases, after which the decline continued as rapidly as before.

In Moray and Nairn, there were three distinct peaks. The first in the thirty-third week, corresponding with the first peak in Glasgow and Ayrshire. The second in the thirty-sixth and thirty-seventh weeks, corresponding with the peak in Edinburgh and the latter part of the Ayrshire summit. The third corresponds with the peak in Dumfries-shire in the fortieth and the forty-first weeks, and therefore with the arrest in the decline in Glasgow.

After the forty-third week, notifications in all five areas continued steadily at a low level until the end of the year.

There can be no doubt that the prevalences in the several areas under consideration were related. The possible nature of this relationship will be discussed later.

The relationship between the industrial and rural communities is fairly well-marked. Glasgow and Ayrshire showed simultaneous sharp rises in the twenty-ninth to thirty-third weeks. Edinburgh followed a fortnight later. Dumfries-shire in the south and Moray and Nairn in the north showed slower rises, culminating in peak incidences when the decline in the other areas was imminent or actually under way. This is clear evidence of the peripheral spread from the main centres of infection.

#### Clinical Material.

Up to the end of August, 1949, the investigation has covered a total of 117 cases. These fall into the five main groups indicated below.

1. Cases of paralytic poliomyelitis.....	35
2. Cases of non-paralytic poliomyelitis.....	42
3. Cases of abortive poliomyelitis.....	7
4. Cases proved not to be poliomyelitis.....	19
5. Cases where the final diagnosis was doubtful	14

Total 117

The individual case histories, in summary form, have been placed in an appendix, so that main thread of the investigation should remain unbroken. Each case is identified by a serial number, which indicates the order in which the Health Department received information concerning them. Generally, the serial numbers indicate the order of onset, but there are several exceptions to this general rule. Abortive cases have been identified by the serial number of the main case with which they are related, and a letter of the alphabet.

In considering the data provided by the clinical material, an attempt has been made to work in the logical order, known to unknown. To this end, therefore, the first group of cases to be considered was that in which there could be the least doubt of the diagnosis - those in which paralysis had occurred. The symptomatology of these cases is presented in some detail, and the information extracted used as a terminus a quo for the examination of the other groups of cases. As no specific test for poliomyelitis exists, it has been considered essential to justify the diagnosis by reference to the general pattern.

To save repetition, figures for four out of the five groups have been combined in one table. In some of the cases, examination was not complete, and records do not therefore cover every sign and symptom in the list. The figures given are percentages of cases showing abnormal findings, in which the particular sign or symptom was sought.

#### Symptomatology in Paralytic Cases.

The signs, symptoms and findings on examination of cases in this group which are shown to be of especial significance are as follows:-

Pyrexia,  
Rigidity of neck,  
Headache,  
Muscular pains and tenderness,  
Rigidity of spine,  
C.S.F. lymphocytosis,  
C.S.F. raised pressure,  
Anomalous tendon reflexes.

The frequency of abnormal findings in other examinations is less than 50%. By comparison, the cases in which a diagnosis of poliomyelitis was rejected show significantly lower rates of abnormality in all directions except pyrexia and headache. This variation remained significantly lower in certain of the abnormalities found in less than 50% of paralytic cases. Urinary symptoms, mental functions and vomiting/



Table to Indicate the Percentage Frequency of Occurrence of Symptoms in Cases in which a Diagnosis of Poliomyelitis was Entertained.

	Paralytic	Non-Paralytic	Doubtful	Not Poliomyelitis
<u>Subjective</u>				
Headache	73.	88.1	92.	75.
Muscle Pain or Tenderness	70.	40.5	43.	26.
Vomiting or Nauseas	47.	45.3	57.	26.
Photophobia	9.	16.7	14.	5.
Vertigo	6.	26.2	0.	0.
<u>Objective</u>				
Pyrexia	87.5	83.8	100.	75.
Biphasic	13.	11.6	14.	0.
Rigidity - Neck	78.	87.2	78.	37.
- spine	66.5	64.3	36.	5.
- other muscles	15.	4.8	0.	5.
Head Retraction	6.	16.7	0.	5.
Kernig's Sign	3.	16.7	14.	5.
C.S.F. - Cells	60.	53.9	0.	17.
- Protein	40.	34.5	0.	17.
- Pressure	56.	41.4	17.	16.
Reflexes - Tendon	51.5	57.2	36.	10.5
- Cutaneous	19.5	33.4	21.	10.5
Mental } - Drowsy	22.	14.3	0.	10.5
Functions } - Irritable	16.	11.9	14.	10.5
Urine - Incontinent	19.	4.8	0.	0.
- Retention	12.5	4.8	7.	5.
Tremor - Skeletal				
Muscles	19.	4.8	7.	16.
- Nystagmus	3.	2.4	7.	5.
Faucial Angina	19.	11.9	21.	37.

vomiting are in this category. The picture drawn from this information is of an illness which is characterised by the general complaints of pyrexia and headache, and by certain specifically nervous abnormalities such as rigidity of the neck and spine, painful and tender muscles, and anomalous tendon reflexes. The nervous abnormalities are shown to result from definite disease of the central nervous system by the altered nature of the cerebrospinal fluid, and the raised intra-theal tension.

#### Symptomatology in Non-paralytic Cases.

When cases which are not paralytic, and do not become so, are considered in the same way, the significant signs and symptoms are found to be these:-

Headache,  
Pyrexia,  
Rigidity of spine,  
C.S.F. lymphocytosis,  
Rigidity of neck,  
Anomalous tendon reflexes.

Pain and tenderness in muscles, and raised pressure in the theca are of less frequent occurrence. Signs of minor frequency in the mental functions, urinary system and musculature are also substantially less common. This appears to indicate that cases which do not go on to paralysis are of a lower order of clinical severity as far as the degree of central nervous system involvement is concerned. Conversely, the occurrence of muscular tremor, and urinary retention or incontinence tends to align the case with the paralytic rather than the non-paralytic group. There is, in fact, some degree of overlapping between the definitely paralytic and definitely non-paralytic case. My own personal experience (see Case 92) confirms this. I experienced an intense degree of lassitude in respect of standing and walking, and the use of the appropriate muscles. This lassitude could be overcome by mental effort. Once the effort was made the muscles responded normally. A certain amount of muscle pain and tenderness was experienced, and I can quite imagine that if the condition had been rather more severe, the mental effort required would have been beyond me, and I would have had to admit to the onset of paralysis. This personal experience has given me insight into the Kenny doctrine of mental alienation, without persuading me to agree to the pathological background of the Kenny concept.

#### Symptomatology in Cases Proved not to be Poliomyelitis.

The only items in which there was substantial agreement between the accepted cases and those rejected/

rejected were:-

Headache,  
Pyrexia.

Their universality as symptoms throughout the field of medicine needs no reiteration. Quite a substantial proportion of the disproved cases did, however, exhibit definite signs of involvement of the nervous system. An examination of the final diagnoses reached is therefore called for at this juncture.

The first main group of rejected cases consisted of those suffering from definite diseases of the central nervous system. In this group there were three cases (Nos. 76, 91, 107), two of tuberculous meningitis and one of cerebral neoplasm. This group accounts for all the cases in which the constituents of the cerebrospinal fluid was abnormal. The main points which led to the change of diagnosis were as follows:-

#### 1. Duration of symptoms.

In the case of the neoplasm, and one of the cases of meningitis, the history covered a period of several weeks.

#### 2. Failure to Maintain Improvement.

After lumbar puncture all three cases were immediately and substantially relieved, and by the time thirty-six hours had elapsed, all three were as bad as ever. Rigidity of the neck was substantially more pronounced in degree and in one case there was definite retraction. This leads to one observation concerning the rigidity of the neck in cases of poliomyelitis. It is that though the neck is abnormally rigid, this rigidity is not of the severity habitually encountered in cases of meningitis. The head can usually be flexed forward till the chin reaches the chest, whereas in a well developed case of meningitis this cannot be done.

It is worthy of note that both cases of tuberculous meningitis were recognised as not being poliomyelitis before tubercle bacilli were recovered from the cerebrospinal fluid. The initial change in diagnosis in the case of cerebral neoplasm was to one of suspected tuberculous meningitis. This was amended post mortem to one of cerebral neoplasm impinging on the lateral ventricle, and thus producing the cytological and biochemical changes. I have previously stressed the importance of distinguishing at an early date between the phase of cerebral irritation in poliomyelitis and early tuberculous meningitis. Often the only means of differentiation is/

is a careful review of the nature and severity of the neurological findings.

The second main group consisted of infections of the upper respiratory tract. Four cases of tonsillitis (Nos. 12, 98, 105, 108) one case of tonsillitis with otitis media (No. 18), one of otitis media alone (No. 11), and one case dubbed "influenza" (No. 106), have been placed in this group. All had substantial headache and pyrexia, and the throat inflammation led several of the patients to hold their heads stiffly, giving the impression of nuchal rigidity. Most of these cases were admitted in the early stages, before the throat, ear, or general condition had developed, and presented therefore no more than pyrexia, headache, and in three cases vomiting, along with some pain and stiffness in the neck. The diagnostic error is, therefore, comprehensible. It is worthy of note that many of these upper respiratory infections are of streptococcal origin, and that intense headache is often a feature of the onset of a streptococcal infection. All these cases were differentiated shortly after admission to hospital, as a result of examination and observation. There is a tendency for these cases to be reported by two classes of medical practitioner, the one who has recently seen several cases of poliomyelitis in the acute phase and is, as it were, sensitised to the condition, and, on the other hand, the recently qualified practitioner, often a locum tenens, whose examination of cases is rather less thorough, and whose evaluation of symptoms less experienced.

The third group is comprised of those cases in which there is an obvious toxic condition, accompanied by meningeal symptoms. Five cases of this general type are included. They were as follows:-

Gastro-enteritis and meningism (No. 62),  
Teething, (possibly gastro-enteritis) and  
convulsions (No. 56),  
Pneumonia and meningism (No. 71),  
Chickenpox, sepsis, and meningism (No. 88),  
Oophoro-salpingitis with meningeal  
irritation (No. 80).

This group raises the general question of meningism occurring as an apparent complication in a case of illness of an otherwise definite and clear-cut nature, and I propose to deal with it in considering diagnosis and differentiation.

The fourth group, of three cases, contains those with some psychological disorder. One was a hysterical woman, with retention of urine (No. 27). The second was a woman with hysteria, and no physical abnormality whatever (No. 51). The third was a girl of/



of fifteen years, with a poor home background, in trouble and unhappy at her work, whose fibrositis in the neck, of sudden onset, gave the opportunity to avoid work (No. 103). All these cases were readily recognised by means of careful investigation.

The last case, the nineteenth, was, perhaps appropriately one of acute alcoholism (No. 74). In fairness to the practitioner reporting him, it must be stated that the patient was in lodgings where little was known of his doings, and that a full history only became available on his recovery after the appropriate lapse of time. Armed with this, the diagnosis was obvious. Without it, and facilities for careful examination, the practitioner was at a severe disadvantage.

#### Symptomatology in Doubtful Cases.

This group of fourteen cases presented the following main signs and symptoms:-

Pyrexia,  
Headache,  
Rigidity of neck,  
Vomiting.

Muscle pain, spinal rigidity and reflex anomalies were present in from a quarter to half the cases.

The main point in evaluation is that in no case was a pathological cerebrospinal fluid secured. The reason for not rejecting the diagnosis out of hand is that the intercurrent illnesses, from which they suffered, did not account for the clinical conditions.

These cases may be grouped in much the same way as those deemed not to be cases of poliomyelitis.

The first group of three cases consists of those suffering from mild pneumonia in which meningism was a marked feature from the start. Two of these were associated both at school and at home, and the aetiological similarity was striking (Nos. 59, 60). They were boys of 5 years and 6 years respectively, the third was a girl of the same age (No. 65). In addition to headache, pyrexia, and vomiting - occurrences common to poliomyelitis and pneumonia, all had neck rigidity, the two boys spinal rigidity. One boy showed anomalous tendon reflexes, the other, tremor of skeletal muscles. None had any abnormality of the cerebrospinal fluid. In all three, the pneumonic condition rapidly resolved on treatment with penicillin, but the meningeal condition persisted. In fact, they were cases of meningism with pneumonia rather than pneumonia with meningism. For this reason they have been classed as doubtful.

The/

The second group, containing six cases, is made up of those with alimentary disorders and neurological signs.

Three of these cases suffered from infection with specific organisms, one with *Shigella Sonnei* (No. 63), and two - a mother and son - with *Salmonella Typhi* Murium (Nos. 82, 83).

Two of the remaining three were cases of gastro-enteritis with meningism (Nos. 67, 110), and the third, a case of marked constipation and meningism (No. 84).

A seventh case lies on the borders of this group (No. 53). In this case, that of a young woman of twenty-three, acute appendicitis developed shortly after admission to the fever hospital. The admission was arranged on account of signs and symptoms warranting a provisional diagnosis of poliomyelitis, with onset some twenty-four hours prior to the appearance of appendicular symptoms. The matter of urgency was to remove the appendix, and by the time that the patient had recovered from the effects of operation, all signs in the central nervous system had vanished.

Two of the remaining cases were single clinical entities. In the one (No. 66), the symptoms were compatible with a diagnosis of either influenza or poliomyelitis. For some reason no lumbar puncture was done, and this chance of making sure vanished. In face of a non-pathological fluid, the dubiety would have remained. In the other case (No. 64), there was a prolonged history of pyrexia, meningeal irritation and convulsions in a boy in his fifth year. The cerebro-spinal fluid was always normal, and not under increased pressure. He was ultimately investigated in the Royal Hospital for Sick Children, Aberdeen, with negative findings. In discussing this case, Professor Craig considered that the diagnosis of encephalitis was justified, and indicated that he had met it, with an autumnal prevalence, not very infrequently in the north-east of Scotland. At least eighteen months after, I learned that this boy's brother had suffered from an illness, which could have been mild paralytic poliomyelitis, in the three weeks immediately following the patient's removal to hospital.

The final case in this group warranted the regrettably vague title of "Pyrexia of Unknown Origin, with Meningism" (No. 99). Apart from the pyrexia and meningeal irritation every examination was without diagnostic result.

#### Symptomatology in Abortive Cases.

Any discussion of the clinical features of poliomyelitis would be incomplete without a short note on the/

the abortive case. These cases must seldom come within the orbit of the Medical Officer of Health, and this by reason of the very fact that they are cut short. The symptomatology in the cases in this series is therefore described at second hand.

The main justification for deciding that a particular case is one of abortive poliomyelitis is, at present, its occurrence in close association with a known case of the disease. Further, it must coincide in time, as well as place. For this reason it is very unusual that a diagnosis of abortive poliomyelitis can be properly made unless there has been a clear-cut case within the victim's immediate circle, preferably his domestic circle. With regard to the time relationship, the abortive case should coincide to within a day or two of the frank case, or should precede or follow it by ten to fourteen days - the approximate incubation period. These criteria have been followed in connection with the seven accepted cases of abortive poliomyelitis included.

As to symptoms, the very fact that the disease has been cut short before the specific picture of irritation of the central nervous system appears, implies that the symptoms are non-specific, and therefore commonly found in medical practice. This has assuredly been the case in the cases named. Headache, vomiting, feverishness and respiratory catarrh, all of brief duration, cover the whole field. No examinations of the cerebrospinal fluid were made, and by the time the Health Department could take action, recovery had occurred.

Undoubtedly there was a general increase in the frequency of minor ailments in Moray and Nairn during the last days of July, and the month of August, 1947. Every general medical practitioner I have approached has agreed with this statement. The cases have been of the type described - respiratory catarrh, or gastrointestinal upset. It is possible, perhaps even probable, that many of these were, in fact, cases of abortive poliomyelitis, but there can be no proof. And there, until some readily applied test becomes available, the matter will rest.

#### Comparison with Other Series of Cases.

The Symptomatology in various other series of cases has been compared with that in the series described, and the wisdom of the dictum that comparisons are odious has become abundantly clear to me. Howe(1) writes with good reason. "It may already be clear that there is no such nicely defined entity as paralytic poliomyelitis and that each patient presents a melange of signs and symptoms indicating damage to various parts of the C.N.S."

In spite of the foregoing, I have thought it worth-while to outline the salient points of agreement and disagreement between my series of cases and others.

The first point to be made is that there is agreement as nearly complete as possible concerning the high incidence of pyrexia and headache, the signs which are not specific to poliomyelitis.

The second point to be made is that there is always a selection of positive findings in the previously mentioned melange. Thus, pyrexia and headache, coupled with rigidity of the neck and spine in one case is the counterpart of pyrexia and headache, with reduced and unequal patellar reflexes, quadriceps cramp, and lumbar pain in another, or retention of urine in a third.

The third point is that in my series there has been little facial angina. I mention this because I have come to believe that its occurrence is unimportant. Perhaps the unusually fine weather of 1947 had an adverse effect on the naso-pharyngeal pathogens which might normally have seized the chance offered by intercurrent pyrexia.

(1) Vital and Rickettsial Infections of Man. T.M.  
Rivers. P.247.



### Diagnosis and Differential Diagnosis.

The section dealing with symptomatology has indicated that the diagnosis in the individual case rests on examination, observation, experience and judgment. Certain problems of diagnosis have been brought to light. Perhaps the most important of these is the place of laboratory aids to diagnosis, and especially the reliance to be placed on the examination of the cerebrospinal fluid. After that in importance comes the question of meningism. And thirdly, I wish to reiterate the importance of prompt distinction between tuberculous meningitis and all other inflammatory conditions in the central nervous system.

### The Cerebrospinal Fluid in the Diagnosis of Poliomyelitis.

Thecal puncture may yield information concerning the pressure of the cerebrospinal fluid, the quantities of its usual constituents, and the presence of invaders.

In bacterial disease involving the meninges, therefore, examination of the fluid may be the means of reaching an exact diagnosis. This is clearly the case where meningitis is present, and is found to be due to pyogenic bacteria. It is eventually the case in tuberculous meningitis. It is never the case in the encephalitides, lymphocytic chorio-meningitis, or poliomyelitis.

In these conditions, and tuberculous meningitis prior to the substantial multiplication of tubercle bacilli, the nature of the condition must be judged on the cytological, chemical and physical condition of the cerebrospinal fluid.

### Cytological Findings.

Marked and persisting granulocytosis is the hall mark of meningitis due to the pyogenic organisms.

Lymphocytosis is the indicator in the tubercular and viral groups. The only question is the number of cells per cubic unit. Unfortunately, there is no rule for the determination of diagnosis by the quantity of lymphocytes present. One thing is certain; there is always a lymphocytosis in tuberculous meningitis. There is almost always one in lymphocytic chorio-meningitis. There is usually one in poliomyelitis, and there may be one in infectious encephalitis.

### Chemical Findings.

Only the variations in the protein and chlorides need/

need be considered.

In tuberculous meningitis, protein is likely to increase early, and to continue in increased amounts.

In poliomyelitis, it will rise later, often only after the patient's symptoms have died down.

In the remaining conditions, changes in the protein are less consistent.

As regards chlorides, tuberculous meningitis follows the pyogenic meningitides, in that the chloride content tends to fall below 660 mgm. per cent at an early stage. In the other conditions it does not show this characteristic fall to such a degree.

To summarise, therefore, in the group of conditions under discussion, tuberculous meningitis is the only one in which changes in the cerebrospinal fluid are certain, and characteristic. In all the others, they follow the same pattern, suggesting that the same mechanism of reaction may occur in all the conditions. There is, however, nothing to demonstrate which condition is present. Furthermore, the present series of cases, as well as others cited, shows clearly that, in about one case of definite poliomyelitis in every three, there is no change in the cerebrospinal fluid. It may be that withdrawal of fluid every few hours would markedly lower this proportion, but the additional information it would afford is hardly worth the added discomfort to the patient. In my view, therefore, examination of the cerebrospinal fluid can serve to demonstrate that an outbreak of poliomyelitis is being encountered, and that diagnosis on clinical grounds may be made with confidence. Examination of the fluid can serve to confirm the diagnosis in a case of doubt - subject to the exclusion of tuberculous meningitis. If, in a single case, however, examination of the fluid gives normal findings, this is not significant in face of substantial clinical evidence in favour of a diagnosis of poliomyelitis. On the other hand, if normal findings are encountered in the examination of a group of cases, whatever the clinical pictures found, a diagnosis of poliomyelitis must be made only with great caution.

#### Meningism.

This condition has received rather scant attention in the textbooks. The following quotations from "A Textbook of the Practice of Medicine", edited by F.W. Price, Sixth Edition, will serve to emphasise this statement.

On page 45 it is stated - "The reason why it may not be possible to decide on clinical grounds whether meningitis/

meningitis is present or not is because several infective processes, other than that due to the meningococcus, are apt to produce symptoms highly suggestive of meningeal irritation. This state of mental irritation, when due to toxæmia and not due to actual meningitis, has been termed meningism or meningismus. The question whether such a state is entirely toxic, or whether it is due to definite though slight changes in the meningeal tissues which just stop short of an inflammatory exudate, is problematical."

Then on page 1567 it is stated ; "The term "meningism" is used for a group of cases which present symptoms of meningitis and in which no pathological change can be found either in the cerebrospinal fluid, or, if death occurs, in the meninges or cerebral tissue. It is met with in children in association with acute febrile diseases, and is presumably due to the toxin present."

The orthodox view is, therefore, that toxins produced in connection with the principal disease circulate in the blood, and, on reaching the central nervous system, produce signs of irritation without leaving any trace of their passage. I see no reason to doubt that this can, and in fact does occur. The simultaneous occurrence of a round dozen cases of meningism, whose principal illnesses were not severe, with an outbreak of poliomyelitis, makes me wonder whether the orthodox view covers the whole field. It is thought by some that the encephalitis which may occur after the exanthemata, and particularly after vaccination may be due to a virus which has been given an especially good opportunity by the principal condition. May it not be the case that the same applies to the virus of poliomyelitis when it is present in a subject contending with another condition, and that the principal condition raises the reaction to clinical levels, when without such reinforcement its effects would have remained subclinical?

#### Tuberculous Meningitis.

In the period of the outbreak of poliomyelitis, August 1947 to August 1949 inclusive, three cases of tuberculous meningitis have occurred. In two of these, the initial diagnosis, for which admission to hospital has been sought, was poliomyelitis. In both cases the cerebrospinal fluid had been examined and, though pathological changes were present, no tubercle bacilli were found. Consequently no firm diagnosis was made. How qualitative differences in the physical signs in these two cases led to the suspicion that they suffered from tuberculous meningitis has already been told. The history of the third case is tragic. The child was teething and the condition attributed/



attributed to this physiological event. She did not improve and the question of poliomyelitis was raised, but the case was not reported nor hospitalised, an expectant policy being adopted. After a fortnight, a second opinion was obtained and a tentative diagnosis of tuberculous meningitis reached. By the time that admission to a streptomycin unit was possible, the child was comatose, dying a few days later. The history outlined makes me utterly opposed to the retention of suspected cases of poliomyelitis at home. Tuberculous meningitis is sufficiently uncommon for a general practitioner to count himself unlucky if he meets more than two cases per decade. Poliomyelitis is now sufficiently common for him to expect to see perhaps a score of cases in that period. The different degrees of familiarity with the two conditions do not warrant the risk of confusion. All cases of suspected poliomyelitis should therefore be sent immediately to the isolation hospital, so that the medical staff there may be given as much time as possible to arrive at the correct diagnosis. The objection might be raised that an ambulance journey in the pre-paralytic stage might precipitate the onset of paralysis. In this series of cases I have found no evidence to suggest that the risk is serious.

#### Treatment and its Results.

Treatment may be considered in relation to the various stages and types of the disease. From the point of view of those working in the isolation hospital, the general management of the case, irrespective of paralysis, is perhaps the most important facet. Thereafter, the early treatment of paresis is of considerable importance. Some attention will also be given to the treatment of respiratory failure.

#### General Management.

In this series of cases, treatment was on orthodox lines.

The most important consideration was to secure the patient's rest. To this end, as far as accommodation permitted, new cases were admitted to single cubicles. Not only did this measure secure isolation pending diagnosis, but removed the patient, at his most irritable, into quiet surroundings. As the general symptoms subsided, and when the diagnosis was established, the patient could be removed into a larger ward for company and economy of staff. While in the single room, suitable sedatives, according to age and cause of restlessness, were given. In very few cases was a small dose of phenobarbitone insufficient. Where it was inadequate, the cause was almost/



almost without fail muscular pain. This called for substantial doses of narcotics in two adult cases. It is of interest to note that these two adults were delirious at times, and for some days suffered from lack of self control. Adequate medication was, therefore, required for the well-being of others as well as themselves (Cases 37, and 38).

Apart from sedation, the most important general measures concerned bowels and bladder. There was a tendency towards constipation in most cases, which called for laxatives. In a few cases there was urinary incontinence, usually of the overflow type. None of the cases in this series presented urinary signs for more than a day or two, and therefore none required catheterisation. Carbachol by the mouth, in doses of 0.002 Gramme, worked well. The care of the bowels and bladder is of importance in securing comfort, and comfort in securing rest. Any discomfort provokes unrest. Discomfort due to distension of the bowels or bladder may pass unnoticed.

In other respects, general treatment aimed at the relief of symptoms was required.

#### Treatment of Early Paralysis.

In a cookery book of a past generation the advice is given to catch the hare before cooking it. In poliomyelitis it is in the same way essential to locate and define the paralysed muscles before treating them. In most cases the presence of paralysis can be readily detected, but its degree, and the whole range of muscles it has involved, is more difficult of assessment. As medical superintendent of the isolation hospital, I therefore made it a rule that paralytic cases, or cases in which paralysis was suspected, should be notified to the Consultant Orthopaedic Surgeon forthwith. This arrangement proved very happy in its working. It ensured early expert advice on the nature and extent of paralytic involvement, and on the methods of immobilisation to be adopted. I have little doubt that the successful outcome of many of the cases in this series is due to the willing promptitude of our orthopaedic surgeon in visiting our cases, often at great personal inconvenience.

At the request of the orthopaedic surgeon, local heat was freely applied to tender and painful parts. This had a very beneficial soothing effect, and in my view ranked as one of the procedures of major importance in the general management as well as the orthopaedic treatment of many cases. In order that hot packs could be conveniently applied, splints were kept as light and simple as possible compatible with their efficiency.

It is interesting to note that patients suffering from paralytic poliomyelitis usually find their greatest comfort in effective immobilisation. So frequently did I observe this to be the case that I would add as a corollary that persistent discomfort in immobilisation calls for re-examination, with the possibility of undetected paralysis in mind.

The cases in this series were also fortunate in that transfer to an orthopaedic unit did not entail change of surgical specialist. The journey to be made was one of about one hundred and twenty miles. Prior to making this journey, the cases were seen by the orthopaedic surgeon, and if necessary special splinting was applied for the duration. All the cases arrived in good general condition.

#### Results of Treatment in Paralytic Cases.

When the effects of treatment are considered, three main groups of cases emerge. In the first are those terminating fatally, in which treatment was wholly unavailing. In the second, are those in which, while life was saved, the residual paralytic disability has remained uninfluenced by remedial measures. In the third are those in which the paralysis has diminished to a greater or lesser extent. This group embraces cases presenting wide divergences in the degree of recovery.

In the following paragraphs only the cases which occurred in 1947 are considered. The paralytic cases indigenous to Moray and Nairn in 1948 were all of the transient type, and those of 1949 are too recent.

#### Fatal Cases.

Amongst the paralytic cases there were five fatalities. (Cases 5, 30, 43, 50 and 58).

Four of these were due to bulbar paralysis. In these, the disease process interfered with respiration, and later with swallowing and the cardiovascular system. Although the cases were treated in the respirator death ensued on account of this further involvement. In three cases out of the four, the temperature rose sharply immediately prior to death.

Death occurred in the fifth fatal case after many weeks in which extensions of paralysis had taken place. The picture presented was similar to that of Landry's paralysis, and death finally was due to respiratory infection. (Case 43).

#### Cases Uninfluenced by Treatment.

There must be some satisfaction in recording that/

that only two cases fall into this category, and of these two one only partially deserves inclusion.

Case 29, at the time of writing is still in the hands of the orthopaedic surgeon with the extent of the paralysis little changed. This patient's respiratory musculature is impaired, and he is liable to contract respiratory infections.

Case 48 presents severe paralysis of the left lower limb, virtually unchanged by treatment. She also sustained a moderate facial palsy, which has completely recovered.

#### Cases with Substantial Recovery.

This group may be further subdivided into three sub-groups. (1) Cases with total recovery, (2) Cases with substantial recovery, (3) Cases with recovery, but still requiring supports.

##### (1) Total Recovery.

Again, satisfaction must be felt that ten cases can be considered totally recovered.

One of these cases (No. 10) sustained a transient but momentarily severe paralysis of the intercostal muscles, necessitating treatment in the respirator as a matter of urgency. Once the initial crisis was past, his recovery by natural means was rapid and complete.

Four of these cases (Nos. 19, 25, 39 and 72), sustained pareses of the musculature innervated from the brainstem - the muscles of the face, throat and neck. These cases followed the rule of complete recovery.

The remaining five (Nos. 3, 6, 14, 17 and 21), were all cases with involvement of one or both lower limbs.

##### (2) Substantial Recovery.

Four cases (Nos. 22, 26, 36 and 41) fall into this group.

Three of these were cases of paralysis of one upper limb only.

The fourth (No. 26) was a case in which there was, initially, severe palatal paresis, which had recovered considerably before discharge from hospital. Some months later a report of the persistence of this paresis was received.

### (3) Cases Showing Recovery, but Requiring Supports.

Six cases (Nos. 1, 7, 16, 32, 37 and 44) fall into this group.

Case 1 presented involvement of one upper and both lower limbs, and, transiently, of the brainstem. She now walks with the aid of a caliper, the other paresis having largely recovered.

In Cases 7 and 16, there was involvement of one lower limb, which has not recovered sufficiently to warrant dispensing with the supporting appliance.

Cases 32 and 44 presented involvement of both lower limbs and the trunk. Neither has been able to dispense with supporting appliances.

Case 37 sustained extensive paralysis in both upper and both lower limbs. She requires a double caliper and wrist support.

### Summary of Results in Paralytic Cases.

This series is too small to justify conclusions except on very broad lines. Certain suggestions are, however, justifiable.

Firstly, there is confirmation of the observation of others that palsies of the facial and ocular type tend towards complete recovery.

Secondly, there is a suggestion that lower limb palsies tend towards complete recovery, while those of the upper limb do not.

And thirdly, it is clear that involvement of more than one part of the cord implies a lessened chance of recovery.

### Summary of Results in Non-paralytic Cases.

The non-paralytic type of case does not lend itself to follow-up methods in the same way as the paralytic. Information is therefore by comparison fragmentary, and capable of being recorded in very general terms.

Most of the cases had made complete, and apparently lasting recoveries by the date of discharge. One case (No. 41) subsequently developed epileptiform convulsions. This is a known sequel, and I am of the opinion that it is too early yet to say that he will prove to be the only case. In several other cases, it is known that convalescence at home after discharge was prolonged. In my own case (No. 95), I have recorded/



recorded my experiences, which included prolonged mental depression, liability to fatigue, and frequency of migrainous headaches. There is some relationship between migraine and epilepsy, though the former is seldom a precursor of the latter.

#### Conclusions Concerning Clinical Material.

The cases described indicate that a sharp local outbreak of acute anterior poliomyelitis has been suffered in the area of Moray and Nairn. Of the one hundred and seventeen cases considered, a diagnosis of poliomyelitis was reached in eighty-four. As far as possible the standard of diagnosis has been checked by the recorded experience of other observers, and I claim that it has proved reasonably accurate. The high proportion of non-paralytic cases recognised supports this claim. In this connection it is of interest to note that, in Scotland as a whole, four cases in five were paralytic, whereas in this series only two in five were paralytic. In the Danish outbreak of 1934, the ratio was roughly one paralytic case to ten non-paralytic. While I do not claim that every case which occurred was likewise brought to light, I consider it unlikely that some two hundred and fifty non-paralytic cases evaded recognition. As will be mentioned later, I have reason to believe that there was an unusual prevalence of minor illness prior to and during the early stages of the outbreak. If any of these cases had been clearly associated with recognised cases of poliomyelitis, they would in all probability, and subject to the symptoms presented and time of occurrence, have been accepted as abortive cases. But I am quite certain that no substantial number of cases of poliomyelitis escaped recognition. The deduction to be drawn is therefore that, relative to the Danish outbreak of 1934, there was a higher proportion of paralytic cases in Scotland in 1947. It is, however, possible that, in areas other than Moray and Nairn, non-paralytic cases occurred with some frequency but passed unrecognised and unrecorded.

As a reflection on the increased frequency of poliomyelitis in the world I mention in passing that Medin's series of forty-four cases, published less than seventy years ago, firmly attached his name to the disease he helped to elucidate, and thereby assured his acclamation as a pioneer of medicine. The present series of nearly double the number of accepted cases is one of many which have been collected and studied of recent years.

The outbreak in Moray and Nairn commenced, as has already been noted, at about the time of the peak in the populous areas, and continued at its height after the decline had set in elsewhere. The relationship of the local outbreak and the national having been determined, it remains to sort out the components of the former, and to compare the two in all possible ways, particularly with reference to the age and sex of cases, and the time relationship of paralytic and non-paralytic cases.

The onset in the first known case was on 26th July, 1947. From that date until early in the year 1948 there was a continuous stream of notifications. In 1948, there were cases in March, June, August and onwards. In 1949, sporadic cases have occurred from May onwards. The general picture, therefore, is of an initial conflagration of some severity, gradually dying away. It is of interest to note that the process of dying away by degrees has involved fairly steady notifications from November, 1947, onwards, except in the depth of winter. This may be considered evidence in favour of the depression of viral metabolism in the colder months.

The case mentioned above with onset on 26th July, 1947, was the first of a group of cases in the town of Rothes and its environs. Similar groups occurred in the towns of Elgin, Forres and Lossiemouth, and to some extent in their environs. The outbreaks in these four towns are the chief components of the outbreak in the county as a whole, and will therefore receive individual consideration. The apparently unrelated rural cases will thereafter be described, and their significance discussed.

#### Cases in the Town of Rothes and its Environs.

Three cases occurred in the town of Rothes (Nos. 2, 2a, and 3), and four cases in the surrounding country (Nos. 4, 6, 6a, and 17). Two cases were abortive (Nos. 2a, and 6a), two were non-paralytic (Nos. 2 and 4), and three were paralytic (Nos. 3, 6 and 17). Their time relationships will be readily seen on reference to Chart VIII at the end of the volume.

The three cases in the town occurred in two houses approximately opposite each other in the High Street. In the one house there were a non-paralytic and an abortive case (Nos. 2 and 2a), in the other, a paralytic case (No. 3). The latter house was associated with a public garage which carried out a certain amount of retail business, and was therefore almost certainly in contact with the first household. Equally, both households were in contact with many others without apparently infecting them.

The remaining cases occurred at two farms three miles north-east, and south of the town, respectively. At the former there were a paralytic and abortive case (Nos. 6 and 6a), and at the latter a non-paralytic case (No. 4), followed after an interval of four weeks by a paralytic (No. 17). The common factor connecting these two households may have been a sale of work held in connection with one of the churches in Rothes, and widely attended, certainly by persons from both farms, and probably also by persons from the two town houses affected.

The three cases in the town of Rothes represent an incidence of approximately 220 per 100,000. The seven cases in Rothes and environs represent an approximate incidence of 350 per 100,000.

No cases occurred in Rothes in 1948, or 1949 up to date.

#### Cases in the City of Elgin and its Environs.

This group is made up of thirty-four cases, which occurred in all three years under consideration.

In 1947 there were eight paralytic cases (Nos. 5, 10, 14, 16, 19, 25, 26 and 58), six non-paralytic cases (Nos. 28, 40, 52, 55, 57 and 61), and three abortive cases (Nos. 5a, 10a and 10b).

In 1948 there was one paralytic case (No. 79), and twelve non-paralytic cases (Nos. 73, 77, 85, 86, 87, 90, 92, 93, 94, 95, 96 and 97).

In 1949, up to 31st August, three paralytic cases (Nos. 102, 104 and 109) had occurred, along with one non-paralytic case (No. 101).

Chart VIII shows their relative occurrence.

The cases occurred in all parts of the town, though the fact that cases occurred close together in the High Street, and in the Ashgrove, a road on the southern outskirts, suggests that more than one minor epidemiological entity had been encountered.

Only two rural cases are included in the 1947 group (Nos. 16 and 40) and they are linked geographically with the cases occurring in the Ashgrove area of the town. This limitation of rural spread is rather surprising considering its relative frequency in the areas around Rothes and Forres. It may be that more of the cases occurring in the deeply rural areas should be attributed to the Elgin group. The town is the county town, and the shopping centre for a wide rural area as well as for Rothes and Lossiemouth. Its two picture houses draw audiences from far afield. The/



The working population of the shops and offices is similarly drawn from a wide area, including the towns of Rothes and Lossiemouth, which, in this connection are virtually satellites. Confirmation of this view is to be found in the extent of public motor omnibus traffic in the area. The local manager of the company which operates so many of Scotland's rural bus services has informed me that between Elgin and Lossiemouth, 1,200 passenger journeys are made daily in each direction, while between Elgin and Rothes, 500 such journeys are made in each direction every day. Between Elgin and Forres the number of vehicles plying is compatible with 1,200 passenger journeys daily in each direction. Vehicles on this route, however, do not ply only between the two towns, but follow one or other of several long distance routes.

Multiple cases were less in evidence than in the town of Rothes, as in only two households was this feature presented (Nos. 5 and 5a, and Nos. 10, 10a and 10b). These were all in the High Street.

Over the whole period the incidence was at the rate of approximately 280 per 100,000 population. In the year 1947, exactly half the cases occurred giving a rate of 140 per 100,000. The two subsequent years have shown declining rates, 108 and 33 per 100,000 respectively.

#### Cases in the Town of Forres and its Environs.

Nineteen cases were confirmed from Forres and its environs. Eight of these were paralytic (Nos. 30, 36, 38, 41, 43, 44, 48 and 50). Ten of the remaining eleven cases were non-paralytic (Nos. 13, 15, 18, 24, 35, 42, 45, 47, 49 and 54). There was one fatal case of polioencephalitis (No. 31), and no abortive cases were reported in the area.

No cases have been reported from this area since 1947. The onset of the first case was on 23rd August, 1947, and the last on 20th October, 1947, just two months later.

Multiplicity occurred in one household, from which two patients were removed to hospital (Nos. 44 and 45). Case 35 occurred in a Convalescent Hospital where the sister of Cases 44 and 45 was employed in a domestic capacity. The onset of Case 35 was on 19th September, 1947, and of Case 44, 28th September, 1947, that of Case 45 being indefinite, but necessitating her removal to hospital two days after her sister. It is conceivable that the infection was transmitted by the intermediary from the hospital to the two children outside. At the same time it must be clearly recognised that the infection could have reached the farm, where the/



the two cases occurred, independently of the hospital. There was, no doubt, the usual coming and going between the farm and the town three miles away. Equally, there was much more coming and going between the hospital and the town. Further, the medical officer of the hospital, being one of the practitioners of the town, had already been in contact with cases of the disease, and could, in theory, have carried the infection to the patient in hospital. So far in this series I have not indicated any pathways of infection in the community. This is the first instance I bring forward, and I am bound to point out that there is no proof that the infection passed from patient to patient in the way outlined.

Case 31, a fatal case of polioencephalitis, was admitted to hospital on 12th September, 1947. She was employed, inter alia, as a milker for a small dairy herd. The milk from this herd was supplied to one mansion-house and four farm cottages. She first felt unwell approximately three weeks prior to admission to hospital, but continued at work until approximately one week prior to admission, that is to say until 5th September, 1947, or thereby. On 23rd September, 1947, or eighteen days after the last occasion on which Case 31 carried out her dairy duties, Case 36 took ill. She was the daughter of the cook in the previously mentioned mansion-house, and resident there. No other case of illness occurred in the mansion-house, and no case of illness occurred in any of the farm cottages. Here again there is evidence of a possible mode of transmission of the infecting agent, but no proof. Intercourse between the mansion-house and the town of Forres was considerable, and must be deemed a possible alternative mode of transmission.

Two of the cases were members of the personnel of a local service establishment (Nos. 24 and 38). Dates of onset were 8th September, 1947, and 27th September, 1947. The former patient had several times visited Forres during the preceding weeks. The latter had been on ten days leave, returning from Northamptonshire on 24th September, 1947. If therefore the formula  $12.2 \pm 1.1$  be used, this latter patient was probably infected between 14th September and 16th September inclusive. That is to say, he may well have been infected away from Forres. Enquiries were made of the Medical Officer at this establishment, and he was able to say that direct contact between the two men was unlikely. Further, he pointed out that there was no increase in the reporting of minor ailments during the period of the outbreak in Forres, and deduced from that that the one patient was infected in Forres and did not bring the infection to the establishment, while the second was infected while away on leave, and also did not bring the infection into the establishment. The argument is attractive but inconclusive.

A further feature of the group of cases in Forres and its neighbourhood calls for remark. Of the nineteen cases, five occurred in the town, while the remainder occurred in the environs. Four out of these five urban cases were in the first five notifications. The Rural cases therefore followed the urban. Furthermore, they occurred in a wide arc, embracing many square miles, round the southern and eastern sides of the town, with an occasional case to the north. This indication of centrifugal spread in one sector made me suspect that there might be a carrier associated with one of the food delivery vans which are now an essential feature of country life. The fact that several of the cases occurred in considerable isolation gave weight to this suspicion. Enquiry, however, was disappointingly inconclusive. No food van could be singled out as contacting a substantial proportion of the rural cases. And none of the affected households was without substantial contact with the town of Forres in addition to that afforded by delivery vans. As a rule, both parents made two visits each to the town every week. If the children were young they accompanied their parents. If older, while not necessarily accompanying the parents, they usually had substantial contacts with the town, often more than the adults.

The incidence in Forres was equivalent, approximately, to 110 per 100,000 population, and in the area involved, to 190 per 100,000, the service cases and establishment being excluded from the reckoning.

#### Cases in the Town of Lossiemouth.

A most explosive outbreak occurred in Lossiemouth in September, 1947, and no further case occurred in the town until May, 1949. The cases in question were Nos. 21, 22, 23, 29, 32, 33, 34, 39 and 72, in 1947, and No. 100 in 1949. There was a tendency for them to be grouped around the school, but in a small town of not more than 5,000 inhabitants, and one large school, no case could be very far from the school, or, for that matter, from any chosen centre in the town.

The onset of the first case was on 2nd September, 1947. One of the other cases took ill the same day, and the remainder on 5th September, 6th September, 9th September, 10th September, 16th September, 24th September and 26th September respectively.

There was a high proportion of paralysis in the cases occurring in 1947, there being six paralytic cases to only three non-paralytic. This is a reversal of the experience of the county as a whole. The paralytic cases tended to be of a fairly severe type.

Some/

Some weeks prior to the outbreak, one of the medical practitioners of Lossiemouth told me that he had seen several cases in which headache and vomiting had been prominent. All the patients concerned had been children, all had spent a day on the beach in the summer sunshine, and all had consumed ice-cream. This may be evidence of the occurrence of abortive cases in the area. Alternatively, it may only be evidence of the popularity and accessibility of the Lossiemouth beach on a fine summer day. The patients which this particular doctor saw were, no doubt, domiciled in Lossiemouth, but many of the visitors to the beach no doubt came from other parts of the county.

In the groups of cases associated with Rothes, Elgin and Forres, there has been evidence of spread into the surrounding countryside. This did not occur in the area of Lossiemouth. The location of the town, and the nature of its environs no doubt accounts for this. To the north lies the sea; to the east is a considerable belt of woodland; to the south lie a few farms and then the wide cultivated lands of the old Spynie Loch, now drained but unsuitable for building; to the west lies a similar wide expanse devoid of population, the airfield and golf course. Within this perimeter lies the town, a few farms, and the quarters for the personnel of the air station, and within this area the cases were distributed.

In no instance is more than one case known to have occurred in a household in Lossiemouth.

The incidence in Lossiemouth was equivalent to a rate of approximately 180 per 100,000 during the year 1947. The single case in 1949 represents a rate of 20 per 100,000.

#### Cases in Other Parts of the County Area.

Fourteen cases occurred in other parts of the county area. Seven of these occurred in 1947 (Nos. 1, 1a, 7, 12, 20, 37, 46), and the remainder in 1948 (Nos. 69, 70, 75, 78, 85, 86 and 89).

The first case notified was from a farm cottage about three miles to the east of Lossiemouth. One abortive case (No. 1a), and a case next door (No. 7) form a small group. The circumstances are such as to call for detailed description.

Case 1, a little girl of 8 years, left home for her summer holidays on 19th July, 1947. To reach her grandmother's home in Glenrinnies, she travelled by bus to Elgin, took another bus to Dufftown, and thence made her way to Glenrinnies. In making this journey she passed through the town of Rothes. She returned from her holidays on 26th July, 1947, travelling by the same/



same route, again through the town of Rothes. Her mother (No. 1a) accompanied her on both journeys. The day 1st August, 1947, was spent on the beach at Lossiemouth, and the child took ill that evening, and her mother at the same time. The following day, 2nd August, 1947, the child had substantially recovered, and went about her normal avocations. On 3rd August, 1947, she was again taken ill and remained confined to bed at home until her removal to hospital on 6th August, 1947, when paralysis had appeared.

In the farm cottage next door, there lived, amongst others, twin baby girls aged nine months. On 15th July, 1947, the twin who eventually became Case 7, was admitted to the isolation hospital, Elgin, with a diagnosis of pneumonia, which was confirmed. She remained in hospital until her discharge home on 2nd August, 1947. Before the end of July, 1947, her twin sister had taken ill, and was nursed in bed at home. On 5th August, 1947, she was admitted to the isolation hospital, Elgin, with a diagnosis of pneumonia, which was confirmed. She remained in hospital until discharged well on 30th August, 1947. Owing to her illness she was not in contact with Case 1 after the latter's return from holidays on 26th July, 1947. The twin babies were a great attraction to Case 1, however, and she lost no time, on 2nd August, in securing Case 7 to nurse and look after. In fact the two spent much of the day together, and Case 1 was instrumental in bottle-feeding Case 7 on that day. This was the only contact between the two. On 11th August, 1947, Case 7 took ill, and on 13th August, 1947, she was admitted to the isolation hospital she had so recently left, with a diagnosis of paralytic poliomyelitis.

Case 1, therefore, had contact with Case 7, and close contact at that, precisely nine days before the onset of the latter's illness, and after the onset of her own symptoms. She did not have any contact with the twin sister, owing to the latter's illness. Apart from Cases 1, 1a and 7, no other cases were reported from the vicinity for some weeks. Prior to the onset of symptoms in Case 1, there was no known case in the whole of Moray and Nairn. The mother (No. 1a) accompanied her daughter on holiday, and it is reasonable to assume was infected at the same time and place. In theory, they could have been infected before leaving home, on the journey away, while away, on the journey home, or after return. The evidence of commencing prevalence in the town of Rothes suggests strongly that they were infected in its vicinity, and not near home. If that is conceded, then there was no source of infection near home for Case 7, who was in any case isolated up till the morning of 2nd August, 1947. The presumption that Case 1 infected Case 7 is strong, and is strengthened by the non-appearance of the disease in the/



the un-exposed twin. The latter in fact played the part of a control animal in the experiment, and what more suitable animal for the purpose than an identical twin?

I conclude, therefore, that Case 7 developed symptoms on the ninth day after exposure, and developed paralysis on the eleventh day after that exposure. This is the only instance in the series of direct transmission from case to case. It is well to remark certain features of the case. The "recipient" was a child in arms, and the "donor" nursed and fed her. What more intimate contact is there likely to be in human relationships than that between a baby and its mother or attendant. The "recipient" was newly discharged from hospital after an attack of pneumonia. It is reasonable to postulate a depression in resistance as a result of this event. The onset of illness had already occurred in the "donor". Experience of other common virus diseases suggests that infectivity is at its height at the onset, immediately before it, or immediately after. It is fair to assume, therefore, that her infectivity was at its height. All the circumstances therefore seem to have conspired together to make the contact between the two children as effective as possible, with the one at her most infective and the other at her most receptive.

Case 12 was the only one in 1947 to occur on the east bank of the River Spey. He took ill and was admitted to hospital on 24th August. His home is in a rather isolated corner of the county relatively remote from bus routes. This perhaps accounts for the absence of spread in the locality. He is a grand-child in a household where the family is scattered through the country in the pursuit of their daily bread. There was the usual family gathering in the early part of August, 1947. At least three visitors from the Glasgow area were in residence during the first two weeks of August, and a fourth, from Aberdeen, was in residence in the middle of the month, from 11th August to 18th August. The assumption that one or other of these persons was the vehicle by which the infection reached the patient seems justified. At the same time, it is impossible to indicate whether the infection came from Glasgow or Aberdeen. Applying the formula  $12.2 \pm 1.1$ , the patient was infected on August 10th, 11th, or 12th, days common to both contingents. At the same time, in the one accepted case of transmission of infection, the incubation period was shown to be nine days to the onset of symptoms, and eleven to the onset of paralysis. As this case was paralytic from onset, the period used for calculation must be eleven days. This gives infection on 13th August, 1947, by which date only the Aberdeen visitor was in residence.

Case 20 calls for little comment. She was a married woman, resident in the married quarters of a service establishment. This unit is fairly remote from Elgin, or the case might have been considered in connection with those occurring there or in its vicinity. The onset took place on 3rd September, by which date only five cases had occurred in Elgin and area, only one of them being outside the town. No history remotely suggesting contact was obtained, nor was there anything to indicate a possible date of infection. No other case occurred subsequently in her vicinity. She had not been away from her quarters and must therefore have been infected in the area of the county.

Case 37 was the only one from Nairnshire in 1947. She was one of the most severely paralysed with whom we had to deal. Her epidemiological history is sufficiently startling and tragic to be re-told.

Symptoms made their appearance on 26th September, 1947, and she was admitted to hospital forty-eight hours later.

On 18th September, 1947, the patient's sister-in-law, who had died of poliomyelitis in Lanarkshire, was buried in Nairnshire. The patient's brother, husband of the deceased, naturally attended the obsequies, and so did the patient herself.

On 30th September, 1947, the child of the deceased, and niece of the patient, was confirmed to be suffering from poliomyelitis in Lanarkshire.

The man, husband of the deceased, brother of the patient, and father of the child - all sufferers from the disease - was, in my view the carrier of the infection to my patient. What his epidemiological relationship was to the others in his household is a matter of conjecture. It seems not altogether unreasonable to regard him as a primary carrier, responsible for infecting all three cases. He may also be regarded as an intermediary between his wife and his sister, the child having been infected by her mother. In any case he was associated with one fatal case, one severely paralytic case, and one case of unknown severity. I repeat my opinion that he infected his sister. If this be conceded, and that infection took place on the date of the funeral, then symptoms appeared on the eighth day after infection, while paralysis was not clearly made out until the fourteenth day.

Case 46 occurred in a residential school. The term commenced on 26th September, 1947. Part of the school routine during the first three weeks of a term is daily inspection, with ascertainment of temperature. On 5th October, 1947, he was found to be unwell, and pyrexial/

pyrexial. He was forthwith isolated in the school sanatorium. Two days later he was seen by a consultant physician who made a tentative diagnosis of poliomyelitis. The boy was removed to hospital in Aberdeen, where the diagnosis was confirmed.

The patient was in contact with his fellow scholars for ten days. Neither then, nor later was there any suspicion of another case in the school. The taking of temperatures continued, and under the circumstances any elevation would have been immediately investigated, but none was reported.

Inquiry in Aberdeen elicited a curious little story. On 23rd September, 1947, or on a date very close to it, this boy met a friend in the street. On 15th October, 1947, the sister of this friend was admitted to hospital in Aberdeen as a confirmed case of poliomyelitis. She probably took ill a day or two earlier, say 12th October, 1947, that is to say sixteen days after my patient returned to school, and nineteen days after his contact with this girl's brother. It is rather hard to envisage a boy being sufficiently infective to pass the disease to his friend during a casual meeting in the street, so that it developed on the twelfth day, and yet being insufficiently infective to infect simultaneously his sister, who lived under the same roof. She did not develop the disease for a further week. The connection between the cases is hard to see, and yet one is loath to admit that it does not exist.

Cases 69 and 70 occurred in Nairnshire in March, 1948. They presented the usual blank wall so far as the ascertainment of clear epidemiological data are concerned. Neither was in contact with the other, or with a case or contact in another area. The only definite finding was that a few cases of poliomyelitis had occurred in eastern Inverness-shire at the same time.

Cases 75 and 78 occurred in the large village of Fochabers in July and August, 1948. No history of mutual contact could be secured, nor of association with a case or contact outside the area. One of them (No. 75) had undertaken an extensive motor journey in the days immediately preceding the onset of his illness. This aspect of poliomyelitis will be discussed further at a later stage. The patient's travels are mentioned here as indicating that a wide field of contacts existed.

Cases 85 and 86 are of some epidemiological interest. In the first place they are brother and sister. They come of a large family, to which there is an annual addition. This family is one of the problem families of the area. Their home is always a slum/



slum with filthy beds and next to no other furniture. Their clothing is always foul and inadequate. They have not been vaccinated nor immunised against diphtheria. They suffer interminably from infestation with pediculi and sarcoptes. Such a family's exposure to all ordinary infecting agents must be continuous and prolonged.

At the time in question they were resident in part of an isolated, aged, and decrepit farm-house. Their water supply was drawn from a neighbouring burn. This burn, some three miles higher up its course, received the overflow from the septic tanks of a country school serving some hundred and fifty scholars. One of these, at all relevant times, was the brother of Case 16. It is remotely possible that this boy was, for a time, an alimentary carrier, and that the virus was transmitted to the school by him. From the school it may have made its way via the septic tanks, and burn to the patients' water supply. I find it extremely difficult, in terms of present day knowledge, to account for the delay in transmission from the school to the patients. This hypothetical mode of spread is therefore put forward as a very tentative explanation.

Case 89 needs but the briefest mention. He took ill in Aviemore, Inverness-shire, on 12th November, 1948, was admitted to hospital at Grantown-on-Spey, Morayshire, on 14th November, and transferred to hospital in Inverness on 17th November. He was, therefore, epidemiologically, not a Moray and Nairn case, and was in the area for only three days.

#### Areas not Affected.

On the ground that their residents may be more susceptible, some note of the areas not affected, or only by single cases, is necessary. The discussion which has already passed has shown that the main centres were the towns of Elgin, Forres, Lossiemouth and Rothes, with a certain dispersal into the corresponding landward areas. These four towns lie between the River Spey on the east, and the River Findhorn on the west. Up to the end of 1947, one case had occurred east of the Spey, and one west of the Findhorn. Since then, two cases have occurred in Fochabers, east of the Spey, and two in Nairnshire, west of the Findhorn. The hill country to the southward has also been largely free of cases. The one who spent three days in hospital in Grantown-on-Spey can be disregarded. Apart from him, the only two cases in the hill country were in the Rothes group. Virtually, therefore, all the cases have occurred in a coastal strip twenty miles from east to west, and ten miles inland from the sea. This area includes the main centres of population, but does not take in some quite substantial sections of the populace. Furthermore, even/



even in this rectangle, there are substantial pockets as yet untouched. These pockets deserve some notice.

The cases in the Elgin and Lossiemouth groups, coupled with six of those which occurred in isolated areas, were all west of a meridian drawn five miles east of the two towns. This leaves a strip of country approximately four miles wide so far entirely free from evidence of infection. This area includes the villages of Lhanbryde, Urquhart, Garmouth, Kingston, and Mosstodloch.

Similarly, there is an area north-west of Elgin, west of Lossiemouth, and north of the mainroad and railway from Elgin to Forres, which has been free from cases, with the exception of the boy in the residential school. This area includes the burgh of Burghead and the still larger population in Hopeman. A recent case, in 1949, included in the Elgin group, suggests that invasion of this area may be about to commence from the south-east.

The gap between the cases around Forres and those around Elgin remains, and in it lies the village of Alves, the school in which is attended by over two hundred pupils from the surrounding farms.

There are therefore plenty of chances for the involvement of unaffected populations in this area, though the main centres now appear to have been saturated.

Reference to Chart IV at the end of the volume will show visually the areas affected, and so far virgin.

#### Cases in 1948 and 1949.

Some separate notice of the cases which occurred in 1948 and 1949, the years subsequent to the main conflagration, is necessary.

Notice has already been given to the occurrence of two cases in Nairnshire in the early months of 1948, and their relationship with prevalence of the disease in the neighbouring part of Inverness-shire has been postulated. Similarly, attention has been given to the occurrence of two cases in Fochabers in July and August, 1948, and their apparent lack of relationship with other cases has been recorded.

Apart from these four cases, all which came to light in 1948 were in Elgin or its immediate vicinity, and all in 1949 in Elgin or Lossiemouth and their immediate environs. This would seem to suggest that in these two centres the possibilities of reaching new and susceptible victims are not exhausted. In its turn/

turn, therefore, the possibility that there are no further opportunities for spread to susceptibles in Forres and Rothes must be entertained. The incidence of the disease in Rothes and Forres, and their environs, in 1947 was definitely higher than that in Elgin, and slightly higher than that in Lossiemouth. Possibly their higher incidences account for their freedom in the subsequent years.

Another feature of the cases in Elgin which has so far had no counterpart in the other centres is the occurrence of vertigo as a presenting feature of several cases in 1948. This occurrence warrants discussion in detail. Had I not been one of the cases myself I doubt if I would have elicited much of the information now put forward.

The cases in question are Nos. 92 to 97 inclusive. A group of them occurred in May and June, 1947, and two more later in the year. Inquiries amongst the medical practitioners in the area lead me to believe that other similar cases have occurred, but have not been considered to be suffering from poliomyelitis.

Briefly, the symptomatology may be stated to include pyrexia, headache, vertigo, nausea and vomiting, nystagmus, ataxia, anomalies of tendon and cutaneous reflexes, and rigidity, especially of the spine. The patients were all adults, with a substantial majority aged forty years or more. None of these cases was admitted to hospital, and in none was the cerebrospinal fluid examined. I regret that this should have been so, but can understand it quite well. I thought I was contracting influenza, and took what I considered to be appropriate action. It was only after several days that I realised that my condition was not influenza. By the time I secured medical advice improvement was setting in. My fellow sufferers adopted similar methods. Had we been affected a little more than a month later, on or after 5th July, 1948, earlier advice might have been secured. By the time that the two cases, which did occur later, came to light, the relatively benign nature of the condition was recognised. None of us had any usual contacts in our daily lives.

It may be argued that these cases were not poliomyelitis. I have considered the matter carefully, in the light of personal experience, and general experience in the recent outbreak, and have come to the conclusion that, in the present state of knowledge, the diagnosis was justified. Firstly, the clinical appearances definitely indicated an invasive condition, with irritation of the central nervous system. Secondly, vertigo and ataxia apart, the signs and symptoms presented were those of non-paralytic poliomyelitis. Thirdly, the two cases from Fochabers, which/

which occurred slightly later in the year, were admitted to hospital, were deemed to be suffering from poliomyelitis, and had typical reactions in the cerebrospinal fluid. It is insufficient merely to criticise these arguments destructively, an alternative explanation must be produced. I have satisfied myself that none of the well-known conditions other than poliomyelitis can readily give the picture presented, and that the only reasonable alternative is to postulate a new disease. This I am not prepared to do. "Entia non sunt multiplicanda praeter necessitatem."

One problem is raised by these cases, particularly by my own. I was in contact with cases of poliomyelitis almost daily in August, September and October, 1947. Not only were the visited and examined in the isolation hospital, but repeatedly visits were made to patients in their homes to consult with the family physician. I should have had ample opportunity in these months to come into contact with the virus. However, not until June, 1948, did the virus succeed in penetrating my defences. The explanation might be that I was particularly exposed in May and June, 1948, but I find difficulty in accepting this explanation. A second explanation, or perhaps speculation, is that some change in the virus had enabled it to penetrate my defences, and at the same time caused it to produce an atypical attack of the disease.

There may be a connection between the occurrence of cases in 1948, and the weather immediately preceding. The detailed daily variations in temperature, moisture, and sunshine, are not very readily obtainable. A general description of weather conditions must therefore suffice.

The monthly Weather Report of the Meteorological Office indicates that April, 1948, was "a warm and generally sunny month." In Forres, the rainfall for the month was 2 inches, the sunshine 154 hours, and the mean temperature at 46.3°F was 2.6% above the average.

May, 1948, was "changeable, with a notably fine spell around the middle of the month; unusually sunny." The rainfall in Forres was 1.9 inches, the sunshine 228 hours, and the mean temperature at 48.4°F was 0.7% below the average.

June, 1948, was "unsettled, wet and rather cool." The rainfall for the month in Forres was 4.8 inches, the sunshine 149 hours, and the mean temperature 53.5°F was 0.3% below the average.

July, 1948, was "very cool and dull until the 17th; unusually hot and sunny in the last week." The rainfall in Forres was 2.7 inches, the sunshine 161 hours, and/



and the mean temperature 56.9°F was 0.4% below average. Temperatures reached 70°F on several dates between 25th July and 31st July, and exceeded 80°F on 30th and 31st July.

August, 1948, was "unsettled, dull and wet, with heavy local rainstorms." In Forres the rainfall was 5.8 inches, and the sunshine only 80 hours. The mean temperature was 55.1°F, and 2.0% below the average. The warm weather of July persisted until 2nd August.

The foregoing recital of weather conditions serves to throw into relief two very fine periods against a background of cold and rain. They are May 14th to 21st., and July 25th to August 2nd.

Cases of poliomyelitis occurred in the period 20th May to 5th June (Nos; 93 to 97), July 26th, August 7th and 24th., other cases occurred in March, and later in October and November. The small number of cases mentioned may be held to have been related in the time of their occurrence to the two periods of fine warm weather mentioned. The number of cases is inadequate to state a conclusion.

In 1949, cases have occurred at fairly even intervals from May until the end of August. This year has been unusually dry, and remains so. The temperature has been steady throughout the period, but has rarely exceeded 70°F. In this year there have been no clear-out weather features - dryness apart - and no clear-cut relationship between weather conditions and the occurrence of cases.

#### Lines of Infection Leading to Moray and Nairn.

Inquiries revealed that eleven cases in 1947, and two cases in 1948, had come to this county within a few days of the onset of symptoms. Several of these cases were local residents who had been away on holiday in other areas, and who made double journeys within the period. Others were true incomers to the area. Particulars of individual cases follow.

#### Case 1.

This patient travelled from her home near Lossiemouth on 19th July, 1947, to Glenrinnies, Banffshire, returning on 26th July, and with onset symptoms on 1st August. The intervals between the date of onset and the dates of the outward and return journeys are therefore, 13 and 6 days respectively. In theory, the patient could have been infected before leaving home, on the outward journey, in Banffshire, on the return journey or after returning home. The fact that the journey involved passing through R othes suggests that she/



she may have been infected there. There is no evidence of infection in Benffshire.

#### Case 2.

This patient travelled from Rothes to Ayrshire and back on dates not precisely recalled, but approximating to 10th July, 1947 and 15th July, 1947. The onset of symptoms occurred on 2nd August, 1947. The intervals were therefore, of the order of 23 days, and 18 days respectively. This patient's baby daughter was accepted as a paralytic case with onset on 26th July. The intervals being, therefore, 16 and 11 days respectively. I consider that infection in Ayrshire or en route is the likely explanation in view of the known prevalence at the time of the holiday.

#### Case 3.

This patient travelled from Rothes to Aberdeen and back on 2nd August and 4th August respectively. The onset occurred on 6th August. It therefore seems likely that he was infected before leaving home, although cases had commenced to occur in Aberdeen at the time of his visit, and the short incubation period has been accepted in certain cases.

#### Case 5.

This was a fatal case of bulbar paralysis. She travelled to Lanarkshire from Elgin on 28th July, returned on 9th August, and took ill on 10th August. Her date of infection would appear to have co-incided with the journey to Lanarkshire, 13 days before the onset. This case I consider to have been infected in Lanarkshire.

#### Case 10.

This little boy travelled to Aberdeenshire on 15th July, 1947, returned to Elgin on 6th August, 1947, and took ill on 18th August, 1947. An interval of 12 days between his return and the onset of symptoms suggests that he may have been infected after his return home. Infection immediately prior to the return journey is an equally probable event.

#### Case 14.

This boy travelled from Surrey to Elgin on 25th August, 1947, and was admitted to hospital on 26th August, 1947. He had previously been ill on 13th August, this possibly being an instance of illness of infection. He cannot have been infected locally, and must in fact have been infected in Surrey.

#### Case 37.

This/

This woman's tragic case has already been mentioned. She did not travel herself, but her brother did, arriving in Nairnshire from Lanarkshire on 18th September, 1947. The onset of the patient's symptoms occurred on 26th September. The circumstances indicate the brother as the means of transmission, and his home in Lanarkshire as the source.

Case 38.

This serviceman went from the Forres area on leave to Northamptonshire on 15th September, 1947, and returned on 24th September, taking ill on 26th September. He could have been infected before setting out, during his journey, or while on leave in Northamptonshire. The neighbourhood of Forres was already an "infected area" at the time he went on leave, equally, there were cases in Northamptonshire at the time, so this also qualified as an "infected area."

Case 46.

This patient travelled to the Elgin area on 26th September, 1947, and took ill at school on 5th October, nine days later. There is therefore, a strong presumption in favour of his infection in Aberdeen prior to coming to Elgin.

Case 49.

This patient travelled to Aberdeen on 4th October, 1947, returned and took ill on 11th October, 1947. The outward journey was therefore seven days prior to onset. In this case, therefore, infection prior to leaving is to be presumed, rather than infection during his stay in Aberdeen.

Case 75.

This patient was in Dumfries from 4th June, 1948, till 11th July, 1948. On that day she commenced her return journey to Elgin, breaking it at Aberdeen on 15th July. The onset of symptoms took place on 26th July, and infection could therefore have taken place either in Dumfries-shire or Aberdeen. It is of interest to note that she attended a wedding in Aberdeen on 15th July, and was not in perfect health from that date. On balance I consider that this suggests infection prior to the visit to Aberdeen, as it must have progressed for some time before being capable of producing symptoms.

Case 478.

This patient set out on an extensive tour by car of the West Highlands of Scotland on 14th August, 1948. This journey took him through Glasgow, and he returned home/

home on 28th August. Symptoms made their just appearance on 24th August, while he was away, and ten days after he left home. The probability here is that he was infected at home, and the possibility that the date of infection was later.

Thirteen cases, not regarding associated abortive cases, were out of Moray and Nairn, or had clear contact with infective areas, in the period immediately prior to onset. My conclusions are as follows:-

- Case 2. was infected in Ayrshire.
- Case 5. was infected in Lanarkshire.
- Case 12. was infected by contact with Glasgow or Aberdeenshire.
- Case 14. was infected in Surrey.
- Case 37. was infected by contact with Lanarkshire.
- Case 46. was infected in Aberdeen.
- Case 75. was infected in Dumfries-shire or Aberdeen.

In seven instances, therefore, there was clear evidence of importation of the infection. In the remainder, the evidence is inconclusive, or suggests infection within the area of Moray and Nairn.

#### Age and Sex of Cases.

The age and sex of victims and its relationship with the previous experience of the community has been described in the introductory part of this study. In regard to the outbreak in Moray and Nairn the clearest picture will be obtained if the data are presented in tabular form.

Table of Confirmed Cases of Poliomyelitis in Moray and Nairn by Age and Sex.

Age Groups	Paralytic		Non-paralytic		Abortive		Total	
	m	f	m	f	m	f	m	f
Under 1 year	2	2	1	-	-	-	3	2
1 - 4	8	3	3	-	2	2	13	5
5 - 14	2	5	10	6	1	-	13	11
15 - 24	4	3	2	2	-	-	6	5
25 Years and over.	3	3	10	8	-	2	13	13
Total	19	16	26	16	3	4	48	36
Grant-Total	35		42		7		84	

This/

This table shows that male cases preponderated over females in the proportion 4:3. This preponderance is most marked in the age group 1 to 4 years, amongst paralytic cases. Non-paralytic cases show a greater degree of preponderance, and this extends to every age-group except of 15 to 24 years where there is equality. Abortive cases show near-equality, there is a female preponderance in the highest age-group, which tips the scale in favour of an excess of female cases in this very small series of cases.

The age of patients also makes an interesting study. Briefly, half the cases are in the age groups 1 to 4 years, and 5 to 14 years; and nearly one third are in the age group 25 years and over. This latter figure results from the high number of adults in the non-paralytic group.

Comparison with the figures for Scotland as a whole is also best made by converting the figures of local incidence to percentages, and comparing them with those for Scotland. The latter figures are available in the Health Bulletin of the Department of Health for Scotland, VI : 1, page 7, in respect of the period 29th June, 1947, to 15th November, 1947.

Age-group	Scotland			Moray and Nairn.		
	m	f	m & f	m	f	m & f.
Under 1 year	7.5	5.9	6.82	6.25	5.6	5.4
1 - 4	36.2	32.0	34.34	27.11	13.9	21.4
5 - 14	34.2	33.1	33.71	27.11	30.4	28.6
15 - 24	12.2	12.63	12.63	12.50	13.9	13.1
25 years and over	9.9	12.50	12.50	27.11	36.1	30.9

There is a substantial difference between the figures in the age-group 1 to 4 years, where the figures for Scotland exceed those of Moray and Nairn, and in the age-group 25 years and over, where those from Moray and Nairn exceed those for Scotland as a whole. Taking into consideration the fact that the local figures represent under one hundred cases, and that those for Scotland exceed 1,500, the differences between the sexes in the several age-groups is insignificant.

The interpretation to be placed upon these figures appears to be that persons of all ages, in Moray and Nairn, were more susceptible to poliomyelitis than were the inhabitants of Scotland as a whole. The figures for the whole country, however, show a considerable proportion of cases in the higher age groups, unlike, say, the figures for Maltese in Malta already mentioned. Scotland therefore, responded as a largely "unsalted" community, Moray and Nairn responded in the same way, but/



but to a more marked extent.

### Multiple Cases.

Eight instances of multiple cases in the same household are recorded. Their particulars may be summarised as follows:-

1. Case 1 was associated with her mother, Case 1a, one of the abortive group.
2. Case 2 was associated with Case 2a, also abortive.
3. Case 4 was followed by Case 17 in the same farm bothy after an interval of 17 days.
4. Case 5 was associated with her sister, Case 5a, an abortive case.
5. Case 6 was associated with her sister, Case 6a, also an abortive case.
6. Case 10 was associated with Cases 10a and 10b, both abortive cases.
7. Cases 44, and 45, sisters, were admitted to hospital within forty-eight hours of each other.
8. Cases 85, and 86, brother and sister, were admitted simultaneously from the same household.

Multiple cases are therefore not very common, though equally not very rare. Of those which have been recorded, only in two instances was there more than one frank case in the household at once (Nos. 7 and 8 above).

In one instance a second frank case appeared sufficiently long after the removal of the first to render other explanations possible (No. 3 above). Here, the second case might have been infected by the first; might have been infected by a common carrier after the first; or might have been exposed to a different source of infection altogether. The last explanation requires an unlikely degree of coincidence in its support. Its immediate predecessor requires the carrier to have remained infective for a considerable period, apparently without infecting any other cases in the vicinity. In passing, it is of interest to note that the two cases were Irish farm labourers, who had arrived at this farm a few weeks previously. The fact that they were selected by the disease suggests that they may have been even more susceptible than the highly susceptible indigenous population. The explanation that the first case infected the second seems the most likely, and bears out the suggestion of higher susceptibility.

In all the remaining instances, the abortive cases were only brought to light by inquiry. If they, and the doubtful multiple just described are disregarded, we find that two instances of multiplicity occurred in respect of seventy-five households, or in under three per cent.

### Infectivity.

The foregoing section, in which it was shown that under three per cent of households were troubled with more than one case is itself prima facie evidence of a low degree of infectivity in so far as the individual case is concerned.

Great difficulty has been experienced in establishing that any particular case was infected from a particular source. Recapitulation of the instances is necessary, though perhaps tedious:-

Case 1 was held to have infected Case 7 by direct contact.

Case 4 may have infected Case 17, and certainly was in sufficiently close domiciliary contact to have done so.

Case 12 was deemed to have been infected by one of four visitors from infected areas, who stayed at his home during the relevant period.

Case 16, or an asymptomatic carrier from his household may, by means of a contaminated water supply, and at a very remote date, have infected Cases 85 and 86.

Case 31 may have contaminated the milk supply of which Case 36 was a consumer. It is only reasonable to point out that Case 37, the guardian of Case 43, and Case 53 did likewise, and that no further case was associated with these other milk supplies.

Case 35 is connected tenuously with Cases 44 and 45, whose household contained one of the domestic staff of the Auxiliary Hospital in which Case 35 was undergoing treatment.

Case 37 is connected by an intermediary with two cases in Lanarkshire. There is a strong presumption that this person was a carrier, and spread the disease by direct contact.

Case 46 was briefly, and at second hand, a remote contact of a case in Aberdeen.

There were, therefore, only eight instances of connection between definite cases. In two of these, the/

the evidence favours the direct spread of the disease from case to case. In two more, there are adequate grounds for pointing to a carrier, or to one of a group which contained a carrier. In the remainder, possible modes of transmission have been pointed out. The weakness of these hypothetical modes is apparent on the most cursory study of the circumstances. They are only included because in none of the remaining cases was it possible to detect any linkage whatever. A tenuous and even an improbable link is thus thrown into a position of importance.

The conclusion to be drawn from the facts, and speculations put forward, can only be that the case of poliomyelitis is relatively lacking in infective power. This view receives strong support from the fact that during the height of the outbreak in 1947, when a score or more cases were in hospital and new ones being frequently admitted, none of the staff of the isolation hospital - medical, nursing, domestic or outside - was affected by the disease, nor by any symptoms which could be considered as due to it. I myself was the only casualty, and the onset of my illness coincided with that of several other cases in the town, and with a time when there were no known cases in the community, either in hospital or elsewhere.

#### Factors Predisposing to the Onset of Poliomyelitis.

The publications of Russell and Hargreaves have already been mentioned in this connection. To recapitulate briefly, Russell demonstrated a causal relationship between muscular effort, and intramuscular injection, and the onset and severity of paralysis. Hargreaves' work is on the same lines. Perhaps his most striking case was that of the man, determined to carry out a pre-arranged holiday motor journey, who carried this out after the onset of symptoms and contracted fatal bulbar paralysis.

I have been largely unable to find confirmatory evidence in my series. In no instance was the onset of paralysis linked with muscular effort. In one instance, a journey made on the second day after the onset of symptoms was associated with fatal bulbar paralysis. In the two other cases where the patient indulged in travel after the onset, no paralysis ensued. Many of the cases received intramuscular penicillin into the gluteal region. Equally, many of the cases so treated developed no paralysis, and in no case could I determine that paralysis of the gluteal musculature was associated with injection.

The foregoing is not to be taken as a statement of disbelief in the findings of these two observers, but merely/



merely that I have not had the good fortune to secure much evidence on the point. I make this particularly clear, because I have evidence which suggests to me that fatigue may favour the onset of poliomyelitis, paralytic and otherwise, and that in particular, the fatigue of travelling may do so.

The evidence mentioned was in part led in the section dealing with Lines of Infection Leading to Moray and Nairn. In that section the dates on which certain of the cases in the series made journeys from, or too, "infected areas" was noted. The aspect which was brought out here was the contact with the "infected area." In correlating this evidence I noted, at an early date, that in quite a number of instances the patient had travelled during the two or three weeks immediately prior to the date of onset. This led me to inquire further into the incidence of travel, and the results are expressed graphically in Chart IX at the end of the volume. This graph records 33 journeys made by 21 patients. The term "journey" is perforce an elastic one, varying as it does from a day of travel from Surrey to Elgin to relatively short bus journeys within the area of the County. None of the journeys were features of the daily lives of the persons who made them.

Study of the graph shows that a substantial number of journeys was made between the thirteenth and tenth days prior to onset, both days included, and again between the sixth and second days prior to onset, both days included. As this occurs in one quarter of all the patients in the series I cannot but treat it as a fact of significance.

I am disposed to associate the journeys made longer before onset with the date of infection. This supposition tallies fairly well with the theories of others concerning the length of the incubation period, particularly with the formula  $12.2 \pm 1.1$  days.

If the supposition, that travel co-inciding with exposure to infection predisposes to it, is accepted as a hypothetical terminus ad quem, it then follows that the vehicle employed for travel is a place particularly favourable to the passage of infection, or that by travel, the bodily threshold of resistance is lowered, so that an otherwise unrecognisable infection becomes clinically recognised. The latter supposition is the more likely. The most likely effects of travel appear to be excitement and fatigue, both of which are phenomena pertaining to the nervous system. It seems, therefore, not too far-fetched a theory to propound that the excitement or fatigue of travel, co-inciding with infection, predisposes towards an attack of poliomyelitis.

Journeys/



Journeys made in the period immediately prior to the onset are less likely to be associated with the date of infection. Study of the graph will show that the peak is reached two days before the onset. The shortest period of incubation for which I have found authority is three days. I therefore put forward hypothetical explanations for this phenomenon as follows. In the case of journeys made four or more days prior to onset, the arguments already led hold good, and the occurrence could be due to infection and predisposition at that date. Alternatively, the effect of travel, fatigue and excitement is so to alter the physiological reactions of the nervous system that the incubation period is shortened. Thirdly, the effect of travel with its attendant fatigue is to bring to clinical levels a pre-existing infection, in the words of the Ministry of Pensions, the condition is aggravated but not caused by the travel. This last supposition appears to me to be the most likely, in though a naturally short incubation period is a possibility which cannot be entirely ruled out.

To summarise, therefore, evidence has been led to show an association between the date of onset and the patient's travelling. The most probable explanations for the occurrence appear to be conditioning the body to the reception of infection, and, nearer the moment of onset, to aggravate the virus potency for harm vis-a-vis its victim. This conditioning or aggravation is most probably due to the lowering of the resistance of the nervous system to the virus through the agency of fatigue.

### Preventive Measures.

Rhodes, in his article in the Bulletin of Hygiene (22:6), to which frequent reference has already been made, outlines the preventive measures usually deemed appropriate. These he divides into three main groups-Information, Hospital Precautions, Precautions for the Public.

#### Information.

The notification of every case "forthwith on becoming aware" of it, is a legal duty laid upon every medical practitioner. This allows the local health department to set in motion its machinery, firstly with a view to verifying the nature of the condition, and secondly to put in hand such precautionary measures as may commend themselves.

Notification was very prompt and adequate in the 1947 outbreak. The commencement of the National Health Service on 5th July, 1947, tended to make the family physician busier, and to make him overlook the need for notification provided he secured hospitalisation of his patient. The importance of liaison between the family physician, the isolation hospital and the medical officer of health - associated as they are with the three types of authority in the National Health Service - cannot be overstressed.

The local health department must also be alive to the family physician's need for up-to-date information, and keep him informed. Similarly, the national department of health must keep the local health department informed of trends in the national situation. In 1947, this information was kept flowing promptly and smoothly in all directions. The doctors of the area were warned of the increasing prevalence of the disease in other areas, and of the progress of the outbreak in this area. They responded extremely well, and the first case was admitted to hospital within four hours of the first appearance of paralysis. They made extensive use of the medical staff of the health department where diagnosis was in doubt, thereby lessening their own burdens of responsibility and providing the latest possible information to the health department. As a rule, cases were admitted to hospital for diagnosis, and lumbar puncture was then carried out. The excellent regional laboratory service, which has been available in the north-east of Scotland for many years, was at all times available to the doctor in doubt about the patient at home.

Most of the medical profession in the area had seen little of the disease, and that when cases were orthopaedic rather than diagnostic problems. As soon as it became available, therefore, the film entitled "Poliomyelitis,"/

"Poliomyelitis," produced under the auspices of the Ministry of Information was shown to an audience of doctors and nurses from the area. The points in diagnosis stressed in this way has led to a very high standard of early diagnosis. (am inclined to think that the showing of this film was the most valuable "preventive" measure carried out, as it secured early diagnosis and rest of patients, thereby reducing the likelihood of severe paralysis.

#### Hospital Precautions.

Rhodes bases his recommendations on the assumption that cases of poliomyelitis are potentially infective through proplets from the maso-pharynx, or through their faeces.

This involves the use of masks by nurses, the sterilisation of crockery and cutlery, which is routine in the isolation hospital, the destruction or sterilisation of handkerchiefs, and above all, the treatment of faeces. by disinfections, as is practised in connection with enteric.

Notice has already been given to the absence of cases amongst the hospital staff. Equally, no case occurred which remotely suggester spread of infection from the hospital by sewage, flies, or on the persons of the staff, whether this immunity is to be placed to the credit of the precautions or not is difficult to decide. In any case the precautions had to be taken, and must be taken in the future until our knowledge of the condition is more precise.

In theory, every patient should be isolated for a period long enough to cover the longest known period of infectivity, that is to say three months. In practice, a much shorter period is found sufficient. Owing to the shortage of hospital accommodation, cases were sent home in Glasgow after fourteen days, if their clinical condition permitted. In this series of cases, it was deemed proper to discharge a patient home at the end of three weeks, if his medical condition were good enough. The same rule prevailed in connection with the transfer of cases to the orthopaedic unit. In practice, most cases were kept in the isolation hospital for four or five weeks, the non-paralytic to allow of walking under supervision, and the paralytic owing to the delays inherent in effecting transfers.

#### Precautions for the Public.

The imposition of a quarantine of three weeks, as mentioned by Rhodes, proved a council of perfection. The housewife could not allow the household to starve, and therefore required access to shops. Economic difficulties would have arisen had wage-earners been too/

too drastically debarred from work. The general principal adopted was therefore to ask the household to keep to itself as far as possible for the three week period. Visits to others or by others were discouraged. Attendance at the picture-house, church, or other gathering of people was barred. Where any member of the household was engaged in the handling of food, he was debarred for three weeks. Children were excluded from school for three weeks - an embargo of easy attainment.

Certain public undertakings assisted in the avoidance of the assembly of crowds indoors, particularly crowds of children, and the Saturday afternoon children's matinees at the picture-houses were discontinued. The swimming baths closed down. Youth organisations delayed the re-commencement of their winter indoor activities.

The schools presented the usual problem, to close or not to close. The outbreak commenced in the school holidays, and had substantially affected Elgin, Forres and Rothes before the term opened. These centres were therefore, deemed "infected areas" before the opening of the new term, with the assumption that exposure outside the school was as likely as within. They were therefore not closed. In Lossiemouth, nearly a fortnight had passed before the second case of school age occurred, the majority being too young to attend. By that time the risk was clearly passing, and closure was not carried out.

In respect of the country schools I had made up my mind to effect closure if a case of poliomyelitis seemed to make school attendance a hazard. The occasion has not yet arisen.

The occurrence of a single case in a residential school has been noted. A visit was made, and fresh air and cleanliness enjoined on every inmate. A quarantine on outside activities was imposed. Arrangements were made to evacuate the school in the event of further cases, but none arose.

In November, 1948, a non-paralytic case occurred in a residential centre for the training of girls of fifteen to eighteen years of age. The likelihood of a serious outbreak at that time seemed slight. Previous experience in a residential school, and in service establishments suggested that spread in this type of community was slight. The patient was, therefore, removed to the isolation hospital, and an official diagnosis of "influenza" made. This served to allay anxiety, and allowed the training centre to continue its work, which would otherwise have been seriously interrupted/



interrupted by the departure home of the pupils.

In other directions, the usual hygienic precautions were maintained. Water supplies, milk supplies, and food distribution ~~was~~<sup>were</sup> kept under continual scrutiny.

In fact, all the normal precautions were taken, and in 1947, there were over 50 cases. It is not possible to say that the precautions were wholly ineffective, but I have strong suspicions in that direction.

## DISCUSSION.

What has been written in the preceding pages may be described as the evidence in the case. Some of this has been based on the observations of others, some on my own. Some discussion and criticisms have been included in the body of evidence, but this has been in the nature of cross-examination. In this section it is intended, as far as possible, to sum up and bring in a verdict. Clearly, there cannot be one verdict, and the discussion will therefore range over the various aspects of the disease which have been described.

### Aetiology and Epidemiology.

These two aspects of poliomyelitis are so closely interwoven that separate discussion would involve unnecessary repetition.

Much evidence, recorded by others and very widely confirmed by second observations, has been led concerning the infecting agent. This is generally accepted to be a virus, of which there are several strains. In the local outbreak, no virus studies were made. By the time the disease reached Moray and Nairn, the laboratories which handle virological material were already overloaded with specimens. Is there then any evidence on the infectivity or otherwise of the disease to be drawn from local experience? Mention has already been made of Medin's conclusion, based on the study of forty-four cases, that poliomyelitis was communicable. Valid conclusions from this series should therefore be possible.

Let the assumption be made, for the moment, that poliomyelitis is not a communicable disease, and see what follows. The alternatives would be that poliomyelitis was due to a physical or chemical agent, to a fault in metabolism, to dietetic deficiency, to endocrine disorder, to neoplasm, or to a psycho-somatic condition.

A physical or chemical agent, which causes harm to the body is of the order of a poison, or produces its effects by reason of alterations of temperature, pressure, position, or relationship with an electrical or radiological field. The physical conditions need not be considered seriously. The cases of poliomyelitis in Moray and Nairn were not - I state it categorically - associated with the physical conditions enumerated, in such degree as to produce the effects recorded. Only the poison is left.

Poisoning implies the administration of a noxious agent, which is inert in that it does not have the property of multiplying itself as living infective agents do. It has to be administered by the alimentary/

alimentary tract, the respiratory tract, or by passage through the skin. The last-named is the last likely route of administration, except where the parenteral administration of drugs is concerned. It is quite unthinkable that a widespread outbreak of poisoning could occur by this means. The only alternative is that some species of creature, which habitually is a blood-sucker, suddenly, and in strictly limited areas, became able to carry a poison. In view of the armies of biting arthropods - bugs, fleas, lice, ticks, mites, mosquitoes, and - particularly in Scotland - midges, the idea that, after centuries of non-toxicity, one of them suddenly becomes possessed of poison is ludicrous.

Poisoning by the inhalation of gases is far from unknown, as any who have but a little knowledge of war gases or industrial hazards must know. The gases concerned act by asphyxiation, as in the case of chlorine, phosgene or methyl bromide, for example, or exclude oxygen from the blood, as in the case of carbon monoxide, or render the higher nervous centres incapable of function, as in the case of nitrous oxide. All these gases require substantial concentrations to be effective. As regards the war gases, the main problem has always been to secure this concentration, and complicated apparatus has had to be devised for the purpose. The liberation of any gas, under natural conditions, in sufficient concentration, and over a wide enough area, to account for the outbreak of poliomyelitis, is inconceivable.

The ingestion of poison in conjunction with food or drink would be brought to light by the routine inquiry into the water, milk and food supplies of the affected households. In fact, no such source of supply has been indicated by the investigation made.

Passing now to faulty metabolism as a possible explanation, I must record the fact that in no case was there any reason to suspect metabolic disorder. The patients were such as might have been selected by random sampling, and included no case of metabolic disorder. Endocrine disorder is a cause of faulty metabolism, and while there was no evidence of metabolic fault, a fortiori there was no evidence of endocrine disorder.

It might be held that dietetic deficiency would be a more likely occurrence in a random sample, such as has been mentioned. In the event, no case of serious malnutrition was found. With monotonous regularity, the admission register of the isolation hospital records that the patient was well nourished. Cases 85 and 86 were the only ones from the group of "problem" families. While these two children were better nourished at the time of discharge than on admission, they did not show signs of marked malnutrition, certainly/

certainly not to the degree of clinical avitaminosis.

The Kenny Concept of poliomyelitis virtually classes the condition amongst the disorders of psychosomatic origin. In order to reach this conclusion, the known pathology has to be disregarded, and this stands on a solid foundation of proven fact. I am not disposed to disregard the possibility that psycho-somatic factors may precipitate or aggravate poliomyelitis, but in the series I have studied I have found no evidence of psycho-somatic causation.

By a process of reductio ad absurdum, therefore, the alternatives to the hypothesis that poliomyelitis is communicable are ruled out of court. That is, provided occult powers such as the evil eye, adduced as an aetiological agent in my grandfather's case, are also ruled out of court. Let a return therefore be made to the assumption that poliomyelitis is communicable, and an examination of the evidence in favour of this assumption be made.

The characteristics of an infectious disease include exposure to a source, a period of incubation, followed by the reproduction of the disease in the new subject. Many of the recognised infectious diseases occur in clear cut outbreaks of greater or lesser extent. The first occurrence, therefore, that suggests the presence of an infectious disease is the simultaneous occurrence of cases amongst the inhabitants of a locality. The outbreak described in Moray and Nairn, and particularly in the local urban communities in the county, satisfies this condition without any dubiety.

The evidence concerning exposure to infection is definite in respect of Case 7, who was undoubtedly, in my view, infected by Case 1. In respect of Case 37, also there was exposure to one who had been in close contact with a case of the disease. In respect of these cases, therefore, the typical clinical picture followed exposure which would have been sufficient to secure the transmission of other infectious diseases. In a number of other instances, histories of possible exposure, of greater or lesser likelihood, were elicited. In yet another group of cases, evidence was led to show a recent visit to, or residence in an area in which the disease was prevalent. In many cases, however, no evidence of the means by which the disease was spread was forthcoming. On balance, however, the evidence sufficed to prove that case to case, and contact to case transmission could occur, but that a more obscure method was usual.

There is direct evidence concerning the period required for incubation in the two cases already mentioned, Nos. 7 and 37. In the first instance the interval/



interval was nine days, and in the second eight days. Both these periods are within the commonly accepted usual limits, namely seven to fourteen days. Both are less than would be given by the formula  $12.2 \pm 1.1$  days.

Further evidence of the possible length of the incubation period comes from the observations on travel in relation to the onset of symptoms. Here it was pointed out that a substantial proportion of cases had travelled between ten and thirteen days before onset, and the likely explanation was put forward that fatigue assisted infection to become severe enough to be clinically recognisable. It does not follow that infection took place simultaneously with fatigue, but there is a strong suggestion that fatigue marked the commencement of effective invasion. If this be accepted, then in eleven instances invasion commenced between thirteen and ten days before the appearance of symptoms, a finding which agrees well enough with the formula already quoted.

Concerning the reproduction of the appropriate clinical picture, little need be said. The analysis of signs and symptoms has left me in no doubt that I have been dealing with a substantial number of cases of poliomyelitis, and recapitulation of the evidence in detail will serve no useful purpose.

If the communicability of poliomyelitis be accepted and in this respect the evidence of the laboratory workers is overwhelming, then it follows that the route by which the disease reached every patient is by some means demonstrable. In fact it was in this series demonstrable in very few instances. Some consideration must therefore be given to ways and means of spread of infection in general.

In the highly infectious diseases, such as smallpox, it becomes essential for the health authorities to trace the contacts of every person potentially a source of infection. If every contact is identified and protected, the outbreak of the disease collapses.

Where, say, diphtheria is concerned, the occurrence of a case warrants the search for other developing cases amongst his contacts, and also for carriers, who are capable of spreading the infection while remaining unaffected. The suspicion that a carrier is operating in a given community is furnished by the persistent occurrence of cases in it. These cases may be more or less frequent.

Where the meningococcus is normally active, it is carried by a proportion of the population continuously. When the proportion of carriers rises, the number of cases of the disease rises sharply and disproportionately. The/

The identity of the carriers probably changes rapidly, but their effects - cases of the disease in a locality - remain constant.

The nature of the outbreak of poliomyelitis described makes the assumption of carriers, as a means of spread, inevitable. In fact, in one instance there is sufficient evidence to justify indicating one individual as a carrier. In one other instance, the existence of a carrier in a certain group of people may justifiably be assumed. Apart from these instances, the explosive nature of the outbreak in Lossiemouth appears to justify the assumption that the disease was introduced by carriers, possibly by a single carrier. The similar explosive outbreak amongst those who attended a function in Rothes justified the assumption of yet another carrier. In none of the cases quoted is there any evidence that the carrier suffered in any way. It appears likely that these carriers were exposed to hundreds of people, and yet produced only a limited number of cases by direct contact infection. How then were the secondary cases infected, in view of the very limited evidence of spread by contact with a case of the disease? I put forward as a speculation that outbreaks of the disease in a locality previously unaffected are likely to be the work of highly infectious carriers, and that the secondary cases are due to carriers of lower infectivity. To my mind, the initial outbreaks in the two towns mentioned resemble outbreaks of diphtheria, where the accepted mode of spread is by respiratory droplets. The long drawn out prevalence, and its widespread nature, in the towns of Elgin and Forres, and their environs, resembles, in my view, the pattern of the prevalence of dysentery due to *Shigella sonnei*. Can the argument be carried further, then, to the speculation that the explosive outbreak of poliomyelitis results from the activities of the nasopharyngeal carrier, and the prevalence of sporadic cases results from the activities of the faecal carrier or carriers? After examination of the facts I am disposed to accept this as a reasonable speculation.

Other possible means of spreading the infection of poliomyelitis have been mentioned in this study, and require brief notice here. One instance of possible spread from case to case by contaminated milk is mentioned. Also one instance where an obviously polluted water supply might have contained virus was mentioned. Such means of spread favour the dysentery pattern rather than the diphtheria pattern. The occurrences were not at the commencement of the outbreak. The first took place two months, and the second twelve or more months after the commencement of the outbreak. I am inclined to regard these occurrences as museum pieces, out of the everyday order, and thrown into prominence by the absence of obvious means of spread on so many occasions.

Numerous other means of spread are recorded in the literature. To name a few, the infection is held to spread upstream, or downstream, in proximity to railway lines, up wind, or downwind. Apart from the fact that the water flowed downstream, to the point where it was drawn by the affected household, and from the point where the overflow from the school septic tanks entered, I can offer no evidence in favour or against any of these views. It is true that there was a tendency for new cases to occur north of previous ones, and therefore nearer the sea, or downstream. It is equally true, however, that all the lines of spread of infection to Moray and Nairn started from points to the southward.

As regards the pattern of the outbreak itself, it followed the well recognised design of spread from one centre of human population to another, followed by centrifugal spread from the area of the first case in each centre. I have already commented on the different attack rates in the centres affected, and their differing experiences in the subsequent years. It seems to me that a local incidence of 200 per 100,000 indicates the saturation of the affected population. The sooner it is reached, the more rapidly the outbreak dies down. If it is not reached before winter puts an end to the season's cases, then there is a recurrence in the following year.

The reaction of the population of Moray and Nairn to the virus of poliomyelitis corresponded with that which authorities identify with an "unsalted" population. Scotland as a whole had had some prevalence in the three preceding decades, but very little in the years immediately before 1947. It is arguable that the population of, say, seven years of age and upwards should have had some immunity. In fact they do not appear to have had any. This supports the view that a new virus was introduced shortly prior to the outbreak. I have, however, not been able to secure confirmation from virological studies.

Concerning the outbreak in Great Britain as a whole, I consider that the evidence points to a concentration of virus in the heavily populated areas long before the epidemic nature of the outbreak became apparent. Whenever such a winter distribution is present it will be followed by a widespread outbreak over the country at large. There will be spread to the peripheral rural areas, each of which will suffer in its turn. Over a period of years the experiences of these rural areas will become uniform, and equal to those of the community at large. I venture to predict that in the next severe national outbreak, which emanates from the centres of population, the experience of Moray and Nairn will be mild, and that some other area/



area will have the doubtful privilege of maximal involvement.

All through this study, weather as a possible factor in the epidemiology of poliomyelitis has been kept in the forefront. Meteorological evidence falls into two main categories - factors which might influence the onset of an outbreak of epidemic proportions, and those which might influence the onset of the disease in a particular case.

I have already expressed the view that the unusual weather conditions of the years 1946 and 1947 favoured the severity of the outbreak. Support for this view is found in the continual epidemicity of the disease at some point in the colder parts of North America, or Northern Europe. This country has a much milder climate than either of the areas named, but when the weather conditions approach those normally found in the areas of epidemic prevalence, then a severe epidemic is experienced. The matter cannot readily be put to the proof. We shall require in this country the juxtaposition of rain, frost and heat which can only be expected very occasionally, and at the same time a substantial prevalence in the populous centres. If such conditions occur without a major outbreak, or an outbreak of comparable extent occur without these meteorological phenomena, then the theory that weather and epidemics are inter-related will be exploded. Till then I hold by it.

In the study of cases in Elgin in 1948, attention was drawn to the occurrence of cases in groups after the spells of hot weather. I take this as evidence that warm weather will favour the spread of virus in a limited way. This is also evidence in favour of spread by faecal carriers. On the whole, the warmer weather favours the spread of dysentery.

There is no doubt that the onset of wintry conditions brings the season's campaign to a close, albeit perhaps a reluctant one. Where the population is not fully exposed, the virus will lie dormant through to colder months, and makes its presence felt with the return of more congenial conditions. An example of such an occurrence is found in the incidence of poliomyelitis in Elgin in 1947, 1948, and 1949. The cases in the first year were insufficient to "salt" the populace, but continued till late in the year. In May and June they re-commenced, continuing again into the autumn. In 1949, after a winter's inactivity, a few more cases occurred. The numbers in each year have been less than in that preceding, and in 1949 were small. I venture the prediction that the local populace is now "salted", and that further cases here are unlikely in the near future.



### Pathology.

No virus studies were made, and there were no adequate facilities for post mortem examinations. In connection with the interpretation of cerebrospinal fluid findings I have already expressed my views. These may be recapitulated briefly.

Where there is sufficient evidence for a diagnosis of poliomyelitis, whether this be paralytic or not, that diagnosis should be made even if the fluid shows no pathological changes. There is no justification for adding to the burden of suffering of the sick for the sole purpose of confirming what is already evident.

The changes found in the fluid are of the order of a lymphocytic reaction, which may be found in other conditions, and is therefore not specific to poliomyelitis.

Repeated examination of a pathological fluid is essential where a diagnosis of tuberculous meningitis has not been excluded. Careful reassessment of the neurological signs is required when the fluid is not diagnostic, and a difference in the intensity of the neck rigidity may warrant a presumptive diagnosis of tuberculous meningitis. Improvement immediately after thecal puncture followed by a return to the previous intensity of signs is suggestive of the alternative diagnosis.

The burden of the foregoing is that diagnosis must rest on clinical evidence, and must not be dictated by the bio-chemist.

### Clinical Features and Diagnosis.

In the series described, the ratio of non-paralytic cases to paralytic is higher than that for Scotland as a whole. The signs of general illness are similar in the two groups. Signs which may be termed paralytic accompaniments - muscle pains, spasms, interference with bladder control - are fewer. Signs of central irritation are slightly less frequent in the non-paralytic case. In this series the initial respiratory catarrh, so frequently reported by observers in other parts of the world, has been conspicuous by its absence.

In my view, provided that there is no frank paralysis, the diagnosis must be reached by an assessment of many signs and symptoms. Onset of illness with pyrexia and headache is common to many conditions. If there is added to this any one or more of the numerous departures from the normal, which have been described and discussed, a diagnosis of poliomyelitis must be amongst those considered.

### Treatment/

### Treatment and Prognosis.

This study has concerned itself little with the minutiae of treatment. As already remarked there has been little real advance in principle, though much in detail, in a century. Where a diagnosis of poliomyelitis has been made, and no paralysis has set in, every effort must be made to prevent this complication. This is achieved by securing rest to body and mind, and this in its turn is, in my view, most likely to be secured in hospital. Once paralysis has set in, the case must immediately come under the care of the orthopaedic surgeon. No effort must be spared to retain the paralysed part in the optimum position while securing the patient's comfort. Skilled treatment at this stage is an insurance against subsequent deformity.

Having seen the state of patients prior to their transfer to the orthopaedic unit, and their condition on return, I am clear that, while prognosis must be guarded, it is much more hopeful than I had previously understood. Under no circumstances should hope of improvement be given up until long after the initial illness. I should not like to commit myself to a period of time in this connection, but am of the opinion that the orthodox views are unduly pessimistic.

### Prophylaxis.

I have already made it clear that the orthodox prophylactic measures were put in hand, and that they were apparently without effect. There is at present no ready means of determining the presence of virus in an individual. Until such a test is found, the carrier, the real problem, will pass untouched. Segregation of carriers, if it could be effected, would obviously be a prophylactic measure of the first order. Segregation of cases must be continued for the maintenance of public confidence. To my mind, the segregation of household contacts seems to have served little useful purpose.

Russell, Hargreaves, and others have indicated a connection between fatigue and paralysis. My series of cases shows what I believe to be a connection between fatigue and the onset of poliomyelitis. These two findings suggest that case incidence, and the incidence of paralysis, might be beneficially influenced by advising idleness during the time when an outbreak threatened. The public might be advised to forego their holidays, to walk instead of running, to go to bed and seek medical advice if feeling the least unwell. This advice, however sound, would probably not be accepted unless, and until, the public became much more afraid of the disease. Until, therefore, the/

the public becomes seriously alarmed, let us recommend the minimum of restrictions, the resolute pursuit of our daily callings, and the imposition of no measures of control which have no certain and useful purpose.

The ultimate prophylactic measure must lie in the full elucidation of the life cycle of the virus and an attack at its weakest point. Thus, vaccination and the sanitary control of shipping have banished small-pox, the slow-sand filter was the beginning of the end for the enteric fevers, and active immunisation has shown the way to freedom from diphtheria. Will some unforeseen antibody formation in the world of viruses, some means of preventing access to the human, or some means of immunisation through the agency of products of virus metabolism be the solution? Time alone will show. Time and the expenditure of adequate sums on virus research.

Summary.

1. An orthodox, but critical review of the present views and beliefs concerning poliomyelitis is presented as an introduction.
2. Against this background, the outbreak in the County of Moray and Nairn in the years 1947, 1948 and 1949 is described.
3. The chief findings in this review were:-
  - (a) A normal clinical type of case.
  - (b) A limited relationship between cases.
  - (c) An incubation period within the accepted period of 7 to 14 days, where it could be determined or inferred.
  - (d) An associationship between fatigue and the incidence of cases of poliomyelitis, but none between it and the site of paralysis.
4. The evidence concerning the epidemiological and other aspects of the outbreak are discussed in relation to the accepted views on the occurrence, management, and prophylaxis of the disease.



A P P E N D I X .

---

FEMALE. 8 Yrs. PARALYTIC.

Onset

1:8:47. Headache and vomiting.  
Unwell for the next few days.  
6:8:47. Paralysis set in. Reported and  
hospitalised.

On Admission T. 100.2. P. 108. R. 30.

Paralysed -

Right upper limb - deltoid, biceps, brachio-  
radialis.  
Left lower limb - early paralysis throughout.  
All affected muscles tender.  
Rigidity of neck and spine.  
Tendon reflexes equal and sluggish.  
Cutaneous reflexes - plantar, flexor.  
abdominal, sluggish.

C.S.F. Cells - 45 per c.mm.,  
Lymphocytes 90%.  
Protein 45 mgm. per cent.

Progress

Pyrexia lasted for four days.  
10:8:47. Respirations fell to 10 per min., sighing  
Respiratory muscles apparently not affected.  
Paralysis eventually involved -  
Right upper limb, from thorax to wrist.  
Left lower limb in toto.  
Right lower limb in toto, rapidly recovered.  
Ptosis of left upper eye-lid, transient.  
Nervous involvement -  
Cord - all areas.  
Brainstem.

Treatment

Stabilisation of limbs by splinting.  
Hot packs to tender muscles.  
Continuous sedation for the first week.  
19:9:47. Transferred to orthopaedic unit.  
10:6:48. Discharged home with weakness of right  
deltoid and left calf.

Contacts

19:7:47. Travelled to Glenrinn, Banffshire.  
26:7:47. Returned home.  
Her mother (Case 1a) suffered from what is deemed  
to have been an attack of poliomyelitis with  
onset the same day.  
Patient was in contact with Case 7 and infected her.

FEMALE. 32 Yrs. ABORTIVE.

Onset 1:8:47.

Symptoms Headache and vomiting for one day.

Diagnosis This rests on the occurrence of typical initial symptoms simultaneously with the onset of paralytic disease in a close family contact.

MALE 33 Yrs. NON-PARALYTIC.

For details of this case I am indebted to the  
Medical Superintendent, City Hospital, Aberdeen.

Onset

2:8:47. Or about that date, severe frontal  
headache occurred.

Temperature 102°F.

8:8:47. Admitted to Gray's Hospital, Elgin, with  
a diagnosis of acute ethmoiditis.

C. S. F. Under increased pressure.  
Lymphocytes, 92% of 330 cells per c.mm.  
Protein 55 mgm. per cent.

Prior to the completion of this examination, patient  
was seen by the Ear and Throat specialist, who  
rejected the diagnosis of sinusitis, and arranged  
transfer to Aberdeen as a case of pyrexia of  
unknown origin.

On Admission

Afebrile.

Headache and photophobia present.

No abnormalities in the nervous system.

Progress

Recovery was uneventful, discharged 31:8:47.

Diagnosis

Rests on the symptoms and examination of the  
cerebrospinal fluid.

Contacts

Patient's daughter, aged 2 years and 4 months, was  
ill with headache and vomiting a week before this  
patient took ill; that is on or about 26:7:47;  
this has been deemed an abortive case (Case 2a).

Patient and family were on holiday in Ayrshire for  
ten days "early in July," the precise dates not  
being remembered, the return journey may, however,  
be assumed to have taken place between 10:7:47 and  
15:7:47.



CASE 2a.

FEMALE. 2 Yrs. 4 Mths. ABORTIVE.

Onset 26:7:47.

Symptoms Headache and vomiting.

Diagnosis This rests on the occurrence of typical initial symptoms one week before the onset of symptoms in the associated non-paralytic case.

MALE 27 years. PARALYTIC.

Onset.

6:8:47. Headache and vomiting

11:8:47. Pain in back of neck, and lumbar region.  
Reported and hospitalised.

On Admission T. 98.4 P. 80 R. 22.

Rigidity of neck and spine, moderate.  
Reflexes normal.  
No paralysis.

C.S.F. Lymphocytes 80 per c.mm.  
Protein 80 mgm. per cent.

Progress

Afebrile in hospital.  
Pain in left thigh was continuously present.  
Progress in other ways satisfactory.

29:8:47. Allowed up, and on two subsequent days.  
2:9:47. Paralysis of left hamstrings, quadriceps,  
glutei, calf muscles, and tibialis  
anterior.

Recovery from paralysis was rapid, and  
almost complete by date of discharge  
22:10:47.

Paralysis corresponded with involvement of  
lower lumbar and upper sacral segments  
of cord.

Treatment

In addition to general measures, sedation,  
immobilisation of affected limb, and physiotherapy  
were utilised.

Discharged with slight limp on left side.

Contacts

Patient was in Aberdeen from 2:8:47 to 4:8:47  
Movements of other members of household were of no  
apparent significance.

MALE 20 Yrs. NON-PARALYTIC.

Onset

9:8:47. Headache and loss of appetite.  
11:8:47. Light-headed, pain in neck, nuchal rigidity.  
Reported and hospitalised.

On Admission T. 100.4. P. 96. R. 24.

Faucial angina slight.  
Rigidity of neck slight, of spine, absent, pain in neck.  
No headache, no vomiting, no reflex disturbance.

C. S. F. Pressure normal.  
Lymphocytes, 10 per c.mm.,  
Protein 50 mgm. per cent.

Progress

Pyrexia persisted for 48 hours.  
Rigidity of neck and pain in neck persisted for ten days.  
18:8:47. Pain in thighs and stiffness over lumbar spine.  
5:9:47. Pain and stiffness has now gone.  
17:9:47. Discharged, kept in hospital for fear of late onset of palsy.

Diagnosis

Rests on symptoms, pathological cerebrospinal fluid, and the occurrence of a paralytic case (Case 17) amongst his contacts.

Treatment

Sedation and local heat to the painful back and limbs.

Contacts

Patient was a travelling Irish farm labourer, and was resident at this particular farm for 2 months prior to the onset of illness.  
For ten days, commencing three weeks prior to the onset of illness a visitor from Keith, Banffshire, was on the farm.  
10:8:47. Two children from Rothes visited the farm.  
1:9:47. Case 17, also an Irish farm labourer, took ill.

FEMALE 6 years. FATAL PARALYTIC CASE

Onset

10:8:47 Vomiting severe  
12:8:47 Rigidity of neck, difficulty in speaking.  
Reported and hospitalised.

On Admission T 102.2 P. 136. R. 30

Rigid neck, respirations short and difficult,  
difficulty in speaking.  
Bulbar paralysis suspected.

C.S.F. Under increased pressure,  
Lymphocytes 26 per c.mm.  
Granulocytes 4 per c.mm.  
Protein 70 mgn. per cent.

Progress

Pharyngeal paralysis set in, followed by respiratory  
paralysis, and death occurred on 14:8:47 at  
8.15 a.m.

Treatment

In the respirator from 9 a.m. on 13:8:47 until her  
death.  
Atropin was used in an attempt to reduce amount of  
secretions collecting in the throat.  
Nasal feeding was adopted.

Contacts

Patient was in Lanarkshire from 28:7:47 to 9:8:47.  
Her sister aged 13 months, took ill with vomiting  
on 13:8:47 and is deemed to have had an abortive  
attack of poliomyelitis. (Case 5a).



CASE 5a.

FEMALE. 1 yr. 1 mth. ABORTIVE

Onset

10: 8:47

Symptoms

Vomiting and malaise.

Diagnosis

Based on simultaneous occurrence with the principal case - fatal paralytic.

FEMALE. 47 years. PARALYTIC.

Onset

11:8:47 Headache and loss of appetite.  
12:8:47 Rigidity of neck slight. Reported and hospitalised.

On Admission T. 98.4. P. 76. R. 22.

Apart from severe headache and slight nuchal rigidity examination was negative.  
Patient was not fully aware of her surroundings.

C.S.F. Under normal pressure and normal in constitution.

Progress

14:8:47. Patient seemed to wake up, becoming and remaining symptom free till discharge on 20:8:47.

In view of the history elicited after admission, that she had exposed herself to the sun on the afternoon of 10:8:47, the diagnosis was at this juncture amended to one of sunstroke.

Immediately after discharge, patient started to drive a tractor in connection with harvesting operations.

Approximately on 27:8:47 weakness of left quadriceps femoris appeared, verified electrically. A history that the pupils had been unequal prior to admission was now obtained, and the diagnosis re-amended to one of paralytic poliomyelitis, with involvement of lumbo-sacral portion of cord.

Treatment

In hospital, entirely symptomatic.  
Paralysis yielded to physiotherapy, and had disappeared at the end of two month's treatment.

Contacts

20:7:47 approximately, there was a sale of work in Rothas, attended by the patient.

11:8:47 patient's sister also suffered from headache and vomiting, and, in the light of subsequent developments, was deemed to have suffered from abortive poliomyelitis. (Case 6a).

CASE 6a.

FEMALE. 45 Yrs. (Approx) ABORTIVE.

Onset 11:8:47.

Symptoms Headache and vomiting.

Diagnosis The original case was one in which a diagnosis of paralytic poliomyelitis was ultimately reached. In view of this, the occurrence of typical initial symptoms in the patient's sister, on the same day, was held to warrant the diagnosis

FEMALE. 9 Months. PARALYTIC

Onset

13:8:47 Patient was very fretful, and her mother noticed that she was not using her left leg.  
Reported and hospitalised the same day.

On Admission T. 102. P. 126. R. 30

Movements of left leg restricted.  
No other abnormality of the nervous system was found.

C.S.F. Under slightly increased pressure.  
Bloodstained and unsuitable for examination.  
As the diagnosis was quite certain, further lumbar punctures were not undertaken.

Progress

Pyrexia continued for 36 hours after admission.  
Child was teething and fretful for first week.

14:8:47. Transient internal strabismus of right eye.  
This did not recur.

21:8:47. Muscular pain in right arm appeared and persisted the following day, but no paralysis developed.

19:9:47. Transferred to an orthopaedic unit.

By this time the left quadriceps femoris was the only muscle involved; reports from the orthopaedic unit indicate substantial improvement.

The lumbar spinal cord was therefore the main point of attack, with the possibility of transient brainstem involvement.

Treatment

The left lower limb was immobilised, at first with sandbags and later by splinting.

14:5:48 Discharged home able to walk and wearing a back splint at night.

Contacts

2:8:47 Patient was discharged from Elgin Fever Hospital where she had been treated for bronchopneumonia.

Her twin sister was also in hospital for this condition, but was not discharged until after Case/



Case 7's re-admission; that is to say the two children were not in contact after 2:8:47, by which date there had been no known case in the area.

On 2:8:47 and the day following, this Case was nursed by Case 1, who lived next door and was very fond of the baby. I have no doubt that this accounts for the infection of this Case, and will discuss the matter in greater detail later.

CASE 8.

FEMALE. 3½ Yrs. NOT POLIOMYELITIS.

12:8:47. Took Ill.

14:8:47. Reported and hospitalised.

On Admission T. 99. P. 124. R. 28.

No signs referable to the nervous system.

Thick nasal discharge.

Tonsils very enlarged and inflamed.

Right otitis media.

Sarcoptic infestation of skin.

Amended Diagnosis

Tonsillitis and otitis media.

CASE 9.

FEMALE. 12 Yrs. NOT POLIOMYELITIS.

14:8:47. Sore throat, headache, pain in back of neck.

15:8:47. Reported and hospitalised.

On Admission T. 99.2. P. 90. R. 20.

Severe coryza.

Faucial angina.

Both tonsils inflamed, with patches of exudate.

No neurological signs or symptoms.

Amended Diagnosis

Tonsillitis.

MALE 1 Year 4 Months. PARALYTIC

Onset

18:8:47 Vomiting, following by breathlessness.

19:8:47 Reported and hospitalised at 4.0 a.m.

On Admission T. 97.4 P. Uncountable R. 36

Breathing difficult, with cyanosis.  
Intercostal muscles apparently totally paralysed.

C.S.F. Lymphocytes 9 per c.mm.  
Other constituents normal.  
Patient was immediately placed in the respirator.

Progress

19:8:47 Temperature rose to 100.8, and remained slightly elevated for 14 days.  
General condition, colour and pulse improved rapidly. Patient was removed from the respirator after approximately 48 hours' treatment, when there had been substantial recovery of the intercostal muscles, which became complete before discharge. Except for the development of a chesty cough, convalescence was uneventful. Discharged 13:9:47.  
The Distribution of the paralysis indicated involvement of the thoracic cord.

Treatment

As already noted the respirator was required for 48 hours.

Apart from this, routine measures sufficed.

Contacts

Patient, with his mother and two brothers, was on holiday in Aberdeenshire from mid-July to 6:8:47. The two brothers suffered from what were deemed to have been abortive attacks of poliomyelitis commencing on 15:8:47 and 17:8:47 respectively. (Case 10a and 10b).



CASE 10a.

MALE      6 Yrs.      ABORTIVE.

Onset      15:8:47.

Symptoms      Headache and vomiting.

Diagnosis      Justified by the occurrence of typical  
initial symptoms commencing the day  
before the onset in the principal case,  
which was paralytic.

CASE 10b.

MALE.      3 $\frac{1}{2}$  Yrs.      ABORTIVE.

Onset      17:8:47.

Symptoms      Headache and vomiting.

Diagnosis      As in Case 10 a.

CASE 11.

MALE. 4 Yrs. NOT POLIOMYELITIS.

19:8:47. Frontal headache, pain in back of neck,  
vomited.

20:8:47. Pain in the knee joints.

21:8:47. Reported and hospitalised.

On Admission T. 99.2. P. 116. R. 24.

Faucial angina.

Otitis media on right side.

Amended Diagnosis

Otitis media.

MALE      5 Years.      PARALYTIC

Onset

24:8:47      Pain in the neck, weakness of legs.  
Reported and hospitalised.  
Six weeks previously he contracted  
pneumonia and had not fully recovered;  
the date of onset is therefore not  
accurately known.

On Admission      T. 99.8      P. 110.      R. 26.

Rigidity of neck and pain on moving head.  
Faucial angina.  
No paralysis of trunk or limbs, but slight internal  
strabismus.

C.S.F.      Under increased pressure, with normal  
constituents.

Progress

Pyrexia was transient and did not recur after  
admission.  
Convalescence was apparently normal.  
Discharged 17:9:47.  
After discharge, slight weakness of the right leg  
developed, but disappeared with rest and the  
passage of time. This indicated involvement of  
the lumbosacral cord.

Contacts

7:8:47      Patient travelled to Elgin.  
Three different persons from the Glasgow area were  
in contact between 1:8:47 and 18:8:47.



MALE.

25 Yrs.

NON-PARALYTIC.

Onset

23: 8:47. Vomited

26: 8:47. Rigidity of neck, spine and both legs.  
Reported and hospitalised.

On Admission

T. 99.8

P. 96

R. 20.

Slight facial angina.

Rigidity of spine and legs, making it difficult  
to sit up.

Pain in both shoulders and both thighs.

No paresis.

Patellar reflexes unequal.

Abdominal reflexes sluggish.

C.S.F.

Normal findings.

Progress

31: 8:47 All rigidity and pain had passed off.

17: 9:47 Discharged after uneventful recovery.

MALE 4 Years. PARALYTIC

Onset

- 13:8:47 Off colour with headache, possibly illness of infection.  
26:8:47 Rigidity of neck, with pain appeared, ascribed to sunbathing.  
29:8:47 Reported and hospitalised as the symptoms were more severe

On Admission T. 99.6. P. 140. R. 30.

Mild faucial angina.  
Rigidity of neck and spine marked.  
Child very drowsy.

C.S.F. Under normal pressure,  
Lymphocytes 61 per c.mm.,  
Protein 40 mgm. per cent  
Sterile.

Progress

- Pyrexia continued for 24 hours after admission.  
31:8:47 Marked head retraction, Kernig's sign present. Patellar, plantar and abdominal reflexes absent  
Incontinent.  
1:9:47 Movement of head and legs still painful  
Incontinent.  
General condition improving.  
4:9:47 Head retraction a prominent feature.

C.S.F. Re-examined to exclude tuberculous meningitis:-  
Lymphocytes 16 per c.mm.,  
Protein 50 mgm. per cent,  
Sterile.  
6:9:47 Unable to sit up.  
Stiffness of lower limbs noted, due to paresis of both tibiales anteriores and spasm of the calf muscles.  
18:9:47 Able to sit up.  
Condition of legs unchanged.  
9:10:47 Transferred to the orthopaedic unit for physiotherapy, and shortly afterwards discharged home fully recovered.  
Paralysis corresponded to affection of the 2nd sacral segment.

Treatment

The extensive spasm and stiffness of the legs was most/

CASE 14 (Contd.)

most effectively combatted with hot packs.  
22:11:47 Discharged walking well with no supports.

Contacts

Patient, with parents, arrived in Elgin from Surrey  
on 25:8:47, the day before the onset of symptoms.

FEMALE. 32 Yrs. NON-PARALYTIC.

Onset

28:8:47. Headache, pain in neck, faucial angina.  
30:8:47. Pain in left shoulder and left calf.  
Reported and hospitalised.

On Admission T. 99.2. P. 80. R. 20.

Rigidity of neck slight, of spine, absent.  
Pain in lumbar region.  
Patellar reflexes sluggish, others normal.

C.S.F. Under normal pressure.  
No cells.  
Protein 40 mgm. per cent.

Progress

This patient was inclined to be hysterical.  
Pyrexia ceased on the day after admission, and  
re-appeared in the second week when other  
symptoms and signs also returned.  
Rigidity of neck, and muscular pains in second  
week.  
20:9:47. Discharged home after satisfactory  
recovery.

Diagnosis

Rests on clinical grounds - pyrexia, rigidity, and  
muscle pain.  
Cerebrospinal fluid protein would be considered  
excessive by some.

Contacts

None to note.



MALE. 16 Years. PARALYTIC

Onset

1:9:47 Pain in back and legs.  
2:9:47 Reported and hospitalised.

On Admission T. 100. P. 88. R. 20.

Headache severe.  
Rigidity of neck not present.  
Rigidity of spine moderate.  
Pain in left calf.  
Patellar reflexes sluggish, abdominals absent.  
Lumbar puncture attempted, but the needle broke and had to be excised.

Progress

Pyrexial for five days, biphasic effect.

4:9:47. Movement of left lower limb impaired;  
flexion and extension impossible, lateral movement slight. The muscles involved were as follows -  
1st lumbar segment Quadratus lumborum  
3rd lumbar segment ilio-pspas, adductors, sartorius.  
4th lumbar segment Abductors, quadriceps  
5th lumbar segment Flexors of knee  
1st sacral segment Muscles of Calf  
2nd sacral segment Glutei, peronei, tibialis anterior.

6:9:47 Apyrexial  
9:10:47 Transferred to orthopaedic unit.

Treatment

5:9:47 Left lower limb splinted.  
23:6:49 Discharged home with long calliper splint.

Contacts

None to note.

MALE. 23 Years. PARALYTIC

Onset

1:9:47 Headache, pain and stiffness of legs after work.

4:9:47 Reported and hospitalised.

On Admission T. 100.4. P. 80. R. 22.

Headache slight.

Faucial angina slight.

Rigidity of neck and spine slight, with pain.

Patellar reflexes absent, others apparently normal.

C.S.F. Under normal pressure,  
Lymphocytes 80 per cent of 36 cells per  
c.mm.  
Protein 60 mgms. per cent.

Progress

Afebrile after the first 24 hours.

Headache and rigidity of neck and spine passed off in three days.

5:9:47 Slight paralysis in the left calf.

Though this was transient, pain in the left calf was very persistent.

Convalescence otherwise uneventful, discharged

4:11:47.

The case was one of minimal paralysis involving the 1st sacral segment.

Treatment

Hot packs to the left calf, otherwise routine measures sufficed.

Contacts

This was the second case from the same farm bothy, the first, Case 4, having been taken ill on 9:8:47.

FEMALE. 30 Yrs. NON-PARALYTIC.

Onset

31:8:47. Headache, nuchal rigidity, loss of  
appetite, vomited.  
4:9:47. Medical advice secured.  
Reported and hospitalised.

On Admission T. 100. P. 100. R. 22.

Frontal headache severe.  
Rigidity of neck and spine.  
Pain in right side of neck.  
No reflex disturbance.  
In the early weeks of pregnancy.

C. S. F. Under normal pressure, with normal  
constituents.

Progress

Afebrile after admission except on isolated  
occasions.  
Asymptomatic after the first week.  
20:9:47. Discharged after normal convalescence.

Treatment

General measures with sedation sufficed.

Diagnosis

Rests on evaluation of the symptoms alone.

Contacts

None to note.

FEMALE. 6 Years. PARALYTIC

Onset

2:9:47 Headache, vomiting, pain in the back of  
the neck and legs.  
4:9:47 Reported and hospitalised.

On Admission T. 102.6. P. 126. R. 36.

Rigidity of neck and spine marked.  
Patellar reflexes exaggerated, abdominal reflexes  
absent.  
Faucial angina present.

C.S.F. Pressure normal.  
Constituents normal.

Progress

The pyrexia was of the biphasic type, falling to  
normal and rising again on 7:9:47, and being  
normal thereafter.  
Mental confusion and delirium persisted during the  
first 4 days.  
7:9:47. Nasal speech, with immobility of the soft  
palate made their appearance.  
11:9:47. Speech and soft palate now normal.  
Convalescence was thereafter uneventful, discharged  
1:10:47.  
Transient brainstem involvement was indicated.

Contacts

None to note.



FEMALE. 25 Yrs. NON-PARALYTIC.

Onset

3:9:47. Headache severe with vomiting.  
5:9:47. Reported and hospitalised.

On Admission T. 102.4. P. 100. R. 24.

Headache severe.  
Rigidity of neck and spine.  
Pain in neck and lumbar region.  
Patellar and ankle reflexes exaggerated, remainder normal.

C. S. F. Under normal pressure with normal constituents.

Progress

Afebrile after three days.  
Nausea was troublesome but vomiting avoided.  
9:9:47. Transient icterus.  
27:9:47. Discharged after satisfactory recovery.

Diagnosis.

Rests on evaluation of symptoms alone.

Treatment

On general lines.  
The jaundice yielded promptly to magnesium sulphate.

Contacts

Resident in the married quarters of a service establishment, from which no other cases were reported.

MALE. 4 yrs. PARALYTIC.

Onset

2:9:47. Headache severe.  
6:9:47. Reported and hospitalised at 10.30 p.m.

On Admission T. 103. P. 122. R. 24.

Frontal headache, pain in back of neck, and, by morning, photophobia, were marked.  
Rigidity of neck slight.  
Rigidity of spine not noted.  
Reflexes presented no obvious abnormality.

C. S. F. Pressure slightly increased,  
Lymphocytes 30 per c.mm., Protein 40 mgm.  
per cent.

Progress

Pyrexia continued for four days.  
7:9:47 Generalised twitching, recurred the following day.  
8:9:47 Retention of urine, followed by incontinence for several days.  
10:9:47. Rigidity of spine became marked, and persisted.  
Generalised paralysis of both legs observed.  
Recovery from paralysis was evident before transfer to the orthopaedic unit on 9:10:47.  
Convalescence was otherwise normal, and the patient was discharged home, with almost complete recovery, one month after transfer to the orthopaedic unit.  
The paralysis corresponded to involvement of all segments of the cord from the 3rd lumbar to the 4th sacral.

Contacts

None to note.

MALE. 1 yr. 11 mths. PARALYTIC.

Onset

- 5:9:47. Not well, no particular symptoms,  
constipated.  
9:9:47. Unable to use the right upper limb;  
reported and hospitalised.

On Admission T. 98. P. 100. R. 36.

Not very ill, no headache, vomiting or rigidity.  
Impairment of movement of right upper limb, mainly  
due to weakness of the deltoid, but also to weak-  
ness of neighbouring muscles.  
Patellar reflexes absent, others present and normal.

C.S.F. Under normal pressure and bloodstained.  
Re-examination gave normal findings.

Progress

Apyrexial during stay in hospital.  
Marked pain occurred in the muscles in the neighbour-  
hood of the right deltoid, with transient pareses.  
Recovery had begun before transfer to the orthopaedic  
unit on 9:10:47.

Final assessment of paralysis -

- 5th Cervical segment - Deltoid, rhomboids, biceps.
- 6th Cervical segment - Deltoid, pectoralis major.
- 7th Cervical segment - Triceps.

When discharged home, early in 1948, partial impair-  
ment of the deltoid alone remained.

Treatment

Sedatives required to be pressed in the early  
stages on account of the pain.

The affected limb was splinted.

General measures sufficed for the rest.

1:2:48. Discharged with complete recovery except  
in right deltoid.

Contacts

None to note.

FEMALE. 12 Yrs. NON-PARALYTIC.

Onset

Not well for a week prior to admission.

8:9:47. Vomited.

9:9:47. Woke at 2.0 a.m. with intense headache and vomiting.

Reported and hospitalised.

On Admission T. 100.4. P. 150. R. 28.

Petechial rash, probably due to dirt and infestation.

Frontal headache intense.

Rigidity of neck and spine.

Kernig's sign present with head retraction.

Patellar reflexes diminished.

C. S. F. Under increased pressure.  
Lymphocytes 90% of 69 cells per c.mm.  
Protein 40 mgm. per cent.

Progress

Temperature fell to subnormal, and rose to normal after 4 days, that is, slightly biphasic.

10:9:47. Headache and photophobia marked.

Irritable and drowsy.

11:9:47. Improved, spine rigid.

11:10:47. Discharged after satisfactory convalescence.

Diagnosis

Rests on clinical and pathological findings.

Treatment

Sedation and general measures sufficed.

Contacts.

7:9:47. The day prior to the appearance of definite symptoms, patient and neighbours made a long motor journey.



MALE. 19 Yrs. SERVICE CASE. NON-PARALYTIC.

Onset

8:9:47. Reported sick with headache and vomiting,  
no pyrexia.  
9:9:47. Headache severe, photophobia, lumbar pain.  
Temperature 101.2. F.  
Reported and hospitalised.

On Admission T. 99. P. 70. R. 26.

Frontal headache and photophobia.  
No rigidity, no pain, Kernig's sign absent, normal  
reflexes.

C. S. F. Under increased pressure.  
Lymphocytes 70% of 47 cells per c.mm.,  
Protein 40 mgm. per cent.

Progress

Apyrexial from the day after admission.  
Headache for three days.  
Spinal rigidity appeared late, at the end of the  
first week, persisting for two weeks and delaying  
convalescence.  
25:10:47. Discharged.

Diagnosis

Rests on symptoms and pathological findings.

Treatment

Sedation and general measures sufficed.

Contacts

One other case (Case 38) occurred in the same  
establishment, but this was presumed to have  
resulted from infection on leave.  
There were no other cases in this establishment,  
and no increase in the sickness rate to suggest  
the occurrence of abortive cases.

MALE. 40 Yrs. PARALYTIC.

There is no first-hand information concerning this patient. Early in August, 1947, he had a bad attack of headache and vomiting, took some whisky, slept for a considerable time, and awoke with a paresis in the left side of his neck. He sought medical advice three weeks later, when paralysis of the platysma was detected electrically. The deeper muscles may have been involved as well, but they are not readily tested. There was no information concerning contacts.

FEMALE. 8½ Yrs. PARALYTIC.

Onset

6:9:47. Headache and faucial angina.  
Pain in back of neck and left shoulder.  
8:9:47. Nasal speech.  
10:9:47. Reported and hospitalised.

On Admission T. 102. P. 102. R. 32.

Rigidity of neck marked, of spine, slight.  
Pain in left shoulder.  
Nasal intonation and difficulty of swallowing.

C. S. F. Under increased pressure.  
Lymphocytes 10 per c.mm.,  
Protein 40 mgm. per cent.

Progress

Pyrexia disappeared in 48 hours.  
11:9:47. Rigidity of spine marked, of neck, still present.  
Patellar reflexes, right - absent.  
left - weak.  
Other reflexes presented no abnormality.  
Difficulty in swallowing was transient.  
Convalescence normal, but nasal intonation still present on discharge on 4:10:47.  
Involvement of the central connections of the motor parts of the 10th and 11th cranial nerves is indicated.

Treatment

At first a close watch for the onset of respiratory paralysis was kept.  
The degree of palatal paresis did not call for special treatment.

Contacts

This child's household received weekly visits from the Grandmother in Lossiemouth.

CASE 27.

FEMALE. 18 Yrs. NOT POLIOMYELITIS.

June, 1947. Appendicectomy.  
August, 1947. Laparotomy, nothing abnormal found.  
10:9:47. Discharged from general hospital.  
11:9:47. Abdominal pain, difficulty in  
passing water.  
Said to have neck rigidity.  
Reported and hospitalised.

On Admission T. 98. P. 76. R. 24.

Abdominal distension marked.  
Neck rigidity, very slight, if present  
at all.  
Retention of urine, catheterized,  
38 fluid ounces withdrawn.  
12:9:47. Stated to have slight spinal rigidity.

C. S. F. Pressure normal.  
Cells 0.  
Protein 50 mgm. per cent.  
17:9:47. Discharged.

Amended Diagnosis

Hysteria, retention of urine.



MALE. 6 Yrs.

*Non Paralytic*

Onset

27:8:47. Headache, possibly illness of infection.

8:9:47. Headache, no vomiting.

Pain in back of neck, back and legs.

10:9:47.

C.S.F. Lymphocytes 75% of 53 cells per c.mm.  
Protein 70 mgm. per cent.

11:9:47. Reported and hospitalised.

On Admission T. 99. P. 112. R. 28.

Rigidity of neck and spine, both slight.

Pain in neck and back.

No reflex abnormality.

Progress

No pyrexia after admission till the third week,  
due to other causes.

Spinal rigidity persisted till 20:9:47.

8:10:47. Discharged after uneventful convalescence.

Diagnosis

Rests on clinical and pathological findings.

Treatment

General measures sufficed.

Contacts

None to note.

MALE. 10 Mths. PARALYTIC.

Onset

6:9:47. Fretful.

10:9:47. Mother noted that the trunk was rigid and that he was unable to get up.

11:9:47. Reported and hospitalised at 1.40 a.m.

On Admission T. 100. P. 180 (?) R. 72 (?)

Rigidity of neck and spine with head retraction.

Arms and legs very stiff, but moved.

Generalised twitching.

Kernig's sign negative.

Patellar reflexes absent, others apparently normal.

The child was very apprehensive.

C. S. F. Under marked pressure.  
Lymphocytes, 51 per c.mm.  
Protein 50 mgm. per cent.

Progress

Pyrexia continued for the first week, markedly biphasic, reaching 104°F. in the first phase and 103°F. in the second.

13:9:47. Both lower limbs flaccid.

Rigidity of spine still marked, and masked the onset of paralysis of the abdominal and dorsal muscles.

Transferred to the orthopaedic unit where he improved slowly.

The paralysis coincided with cord involvement from the mid-thoracic to the mid-sacral segments.

Treatment

After initial sedation, the two legs were immobilised in partial abduction and the child kept recumbent.

17:8:49. Still in orthopaedic unit with paralysis of both lower limbs and intercostals; respiration is maintained by the diaphragm alone; respiratory infection recurs repeatedly.

Contacts

None to note.

MALE. 5 Yrs. FATAL PARALYTIC CASE.

Onset

6:9:47. Suffered from coryza.

10:9:47. Very drowsy and listless, with temperature of 100<sup>0</sup>F.

Pain in right shoulder.

Rigidity of neck thought to be present.

12:9:47. After partial recovery, he was pyrexial, limp, with impaired movement in the lower limbs, and rigidity of neck.

Reported and hospitalised at 5.10 p.m.

On Admission

T. 101. P. 140. R. 30.

It was reported that breathing became difficult in the ambulance, and on reaching hospital, paralysis of the intercostal muscles and diaphragm was noted. He was therefore placed in the respirator at once. Whenever artificial respiration was suspended for nursing purposes, collapse occurred at once. Early the next morning, mucus began to collect in the buccal cavity, suggesting inability to swallow, and the child collapsed and died at 5.55 a.m., just over 12 hours after admission.

Detailed examination of the nervous system was not possible, nor of the cerebrospinal fluid.

The course of the illness indicated severe bulbar paralyses.

Contacts.

None to note.

FEMALE. 50 Yrs. FATAL CASE.  
ENCEPHALITIS.

Onset

This patient was at home for about 3 weeks after the onset, during which she was mentally confused, and suffered from headache, vomiting, and was incontinent of urine.

12:9:47. Reported and hospitalised.

On Admission T. 97.4. P. 70. R. 24.

No paralysis.

No rigidity of neck or spine.

Mentally confused.

Incontinent of urine, skin of back sore, marked pruritus.

Plantar reflexes exaggerated, others not altered.

Progress

Initial improvement for three days.

15:9:47. Spinal rigidity set in.

C. S. F. Under increased pressure.  
Lymphocytes 18 per c.mm.  
Protein 60 mgm. per cent.  
Sterile Wassermann negative.

16:9:47. Pyrexial in the evenings from to-day onwards.

Incontinent of urine and faeces.

Very drowsy and confused.

17:9:47. Marked headache.

Restless and drowsy.

Condition deteriorated in the evening.

T. 98.8. P. 68. R. 16.

Pupils react feebly to light, unequally dilated.

Both upper and both lower limbs moved voluntarily.

Patellar reflexes unequal, ankle reflexes equal.

Urine contained no sugar and no albumin.

C. S. F. Under greatly increased pressure - 240 mm.  
Lymphocytes 7 per c.mm.  
Protein 80 mgm. per cent.  
Sterile.  
After/



After removal of fluid, patient became much more rational.

18:9:47. Became comatose.

19:9:47. Died.

No post-mortem examination made.

### Diagnosis

This rests on history, symptoms, and the pathological cerebrospinal fluid.

The history and symptomatology are consistent with a diagnosis of poliomyelitis.

The cerebrospinal fluid findings are similarly consistent, and are exclusive of septic meningitis, tuberculous meningitis, and syphilitic meningitis.

Lymphocytic chorio-meningitis is not fatal.

The absence of sugar and albumin from the urine exclude uraemia or diabetic coma.

The findings in the cerebrospinal fluid are not entirely exclusive of an intracranial neoplasm, they are, in fact, similar to those in Case 76, but neither cells nor protein are so high as in that case. The absence of epileptic fits and the relatively short history are against this diagnosis.

By exclusion, therefore, diagnosis of encephalitis was reached, and in an area in which poliomyelitis was prevalent, a diagnosis of polio-encephalitis was considered to be justified.

### Contacts

This patient was a milker and drew the milk consumed in the household of which Case 36 was a member.

FEMALE. 1 Yr. 4 Mths. PARALYTIC.

Onset

9:9:47. Vomited and refused food.  
13:9:47. Did not pass urine.  
14:9:47. Unable to sit up.  
Reported and hospitalised.

On Admission T. 98.8. P. 100. R. 28.

Rigidity of neck marked.  
Rigidity of spine not definitely elicited owing to inability to sit up.  
Slight internal strabismus present.  
Patellar reflexes absent, others present and normal.

C. S. F. Under increased pressure.  
Lymphocytes, 98% of 360 cells per c.mm.  
Protein 40 mgm. per cent.

Progress

15:9:47. Passed urine naturally.  
Pyrexia continued, with a maximum of 99.4 F. for three days; at the beginning of the second week it recurred, and persisted two and a half weeks.  
17:9:47. Both lower limbs became flaccid.  
Paralysis rapidly spread and at its height affected the left sternomastoid, the intercostals, muscles of the back, abdominal wall, and both lower limbs.  
9:10:47. Transferred to orthopaedic unit with paralysis nearly maximal, subsequently substantial improvement took place.  
The distribution of the paralysis indicates widespread involvement of the central nervous system; the heaviest weight fell on the lower thoracic, lumbar, and upper sacral portions of the cord; the 2nd cervical segment and the brainstem were also involved.

Treatment

Supine position with legs splinted in partial abduction.  
She was a quiet patient and general measures were otherwise sufficient.  
26:1:49. Discharged home with callipers.

Contacts

One other member of household, a boy aged 4 years was/

CASE 32 (Contd.)

was ill simultaneously with patient, the symptoms were indefinite; this is deemed to have been a case of abortive poliomyelitis. (Case 32a.) An aunt of patient works in the same restaurant as the aunt of another patient, Case 29. The patient, and others in the household, visited Burghead, a few miles away, for a few days - exact number unknown - from 28:8:47.

CASE 32a.

MALE. 4 Yrs. ABORTIVE.

Onset 9:9:47.

Symptoms Indefinite.

Diagnosis Based on simultaneous occurrence with the  
principal case - paralytic.



FEMALE. 8 Yrs. NON-PARALYTIC.

Onset

9:9:47. Headache and vomiting.  
10:9:47. Pain in the back of the neck.  
15:9:47. Rigidity of neck appeared after partial remission of symptoms.  
16:9:47. Reported and hospitalised.

On Admission T. 100. P. 120. R. 32.

Very flushed, pupils dilated.  
Rigidity of neck and spine marked.  
Pain in lower limbs.  
Patellar reflexes absent.

C.S.F. Contaminated with blood and not examined.

Progress

Pyrexia disappeared after 24 hours.

19:9:47.

C.S.F. Under normal pressure.  
Lymphocytes 12 per c.mm.  
Protein 40 mgm. per cent.

Rigidity of neck and spine persisted for about three weeks.

5:11:47. Discharged, the delay was due to inter-current infection of the throat with Vincent's organisms.

Diagnosis

Rests on clinical and pathological findings.

Treatment

No special measures required.

Contacts

Patient was one of several simultaneous cases in Lossiemouth.

MALE. 8 Yrs. NON-PARALYTIC.

Onset

16:9:47. Headache.  
17:9:47. Reported and hospitalised.

On Admission T. 98.4. P. 86. R. 22.

Headache severe, no vomiting.  
Rigidity of neck and spine.  
Head retraction, Kernig's sign present.  
No reflex abnormalities.

C. S. F. Pressure moderately increased.  
Lymphocytes 60 per c.mm.  
Protein 40 mgm. per cent.

Progress

Apyrexial from the time of admission.  
The pulse was irregular during the second week.  
Discharged 25:10:47, after satisfactory convalescence.

Diagnosis

Rests on clinical and pathological findings.

Treatment

General measures sufficed.

Contacts

Other cases in the locality.

FEMALE. 13 Yrs. NON-PARALYTIC.

Onset

In an E.M.S. Hospital for five weeks prior to transfer to the Fever Hospital.

19:9:47. Headache and rigidity of neck.

22:9:47. After remission, these complaints returned. Reported and hospitalised.

On Admission T. 99. P. 78. R. 22.

Headache slight.

Rigidity of neck.

Stiffness of shoulders.

Patellar reflexes - Right, faint

- Left, sluggish.

Other reflexes not altered.

C.S.F. Bloodstained and not examined.

Progress

Afebrile after admission.

Convalescence was satisfactory from the start.

25:10:47. Discharged.

Diagnosis

Rests on symptoms and signs alone.

Treatment

No special measures were needed.

Contacts

This patient was in Altyre E.M.S. Hospital, and is therefore a possible source of infection of Cases 44 and 45, through the agency of members of the household of the latter employed at Altyre.

FEMALE. 5 Yrs. PARALYTIC.

Onset

23:9:47. Severe headache and vomiting, with history of a "chill" one week previously.

24:9:47. Reported and hospitalised.

On Admission T. 100.6. P. 126. R. 28.

The child was in a state of great excitement.

Rigidity of neck and spine were present.

Patellar reflexes - left sluggish.

- right absent.

Plantar reflexes very brisk, others apparently normal.

Incontinence of urine present.

C. S. F.

Under normal pressure.

Lymphocytes, 98% of 172 cells per c.mm.

Protein 50 mgm. per cent.

Progress

Pyrexia persisted for four days and recurred intermittently for the following week.

Incontinence of urine persisted for the first week in hospital.

Pain and rigidity in the neck and back were very troublesome for the first week in hospital.

Marked photophobia was present during the first week.

This child was very restless to begin with.

30:9:47. Restricted movement of the left upper limb due to paralysis of the deltoid and supraspinatus was noted.

This corresponds to involvement of the cervical segments of the cord.

Convalescence was thereafter uneventful, and the patient was discharged home on 5:11:47.

Treatment

During the first week, sedatives were given freely. When paralysis appeared, the arm was fixed in an abduction splint.

Contacts

The patient lived in a large country house, where her mother was cook, but there is no certain evidence of other cases in the household.

The house is supplied with milk from cows milked by Case 31.



FEMALE. 20 Yrs. PARALYTIC.

Onset

- 26:9:47. Headache, pain in back, and rigor.  
 27:9:47. Temperature 101°F. dizzy, lost appetite,  
 nauseated, legs twitching.  
 28:9:47. Severe headache, vomited, rigidity of  
 neck noted.  
 Reported and hospitalised.

On Admission T. 101.6. P. 104. R. 24.

Patient was excitable, complained of severe headache,  
 and exhibited photophobia.

Rigidity of neck and spine present.

Tendon reflexes all sluggish, others apparently  
 normal.

C. S. F. Pressure not raised.  
 Lymphocytes, 98% of 156 cells per c.mm.  
 Protein 160 mgm. per cent.

Progress

Pyrexia of the biphasic type lasted for 4 days after  
 admission.

29:9:47. Generalised stiffness noted.

30:9:47. Joint pains troublesome, other symptoms  
 abating.

1:10:47. Rigidity of spine much more marked,  
 preventing turning in bed.

Both arms and both legs felt heavy, but could be  
 moved.

Lateral nystagmus present.

2:10:47. Restriction of movement of left arm.

Incontinent.

3:10:47. Restricted movement of right arm.

4:10:47. Very restless and noisy this night.

Able to move herself when restless.

Adductor paralysis of the left lower limb, also of  
 flexors of the knee.

10:10:47. The whole right lower limb flaccid.

The muscles eventually demonstrated to be involved  
 were -

Left upper limb - deltoid, triceps,  
 flexors, and extensors  
 of wrist, hand.

Right upper limb - deltoid, triceps.

Left lower limb - Adductors of thigh,  
 flexors and extensors  
 of knee.

Right lower limb - Total involvement.

After/

After transfer to the orthopaedic unit on 4:11:47, substantial improvement in both upper limbs, less in the hand, good improvement in the right lower limb, and less in the left, took place.

The paralysis corresponded with involvement of the four lower cervical segments, and the three lower lumbar and two upper sacral segments of the cord.

#### Treatment

The main feature of this case was the marked cerebral irritation, followed by extensive paralysis of slow development. This permitted dealing with each feature as it arose, firstly, by sedation, and latterly by immobilisation with the patient's co-operation.

24:7:48. Discharged home with double callipers and support for left wrist.

#### Contacts

18:9:47. Patient attended her sister-in-law's funeral, who had died of poliomyelitis in Lanarkshire. The body was brought to Nairnshire for burial.

30:9:47. The dead woman's child took ill with poliomyelitis in Lanarkshire.

The patient's brother naturally accompanied his wife's funeral, and I have no doubt was the means of spreading the disease to this case.

MALE. 22 Yrs. PARALYTIC. SERVICEMAN

Onset

26:9:47. Unwell, no specific symptoms.  
27:9:47. Stiff back, unable to turn in bed.  
Headache commenced.  
28:9:47. Vomited.  
Reported and hospitalised.

On Admission T. 101.2. P. 94. R. 26.

Nauseated, exhibited photophobia.  
Rigidity of neck and spine present.  
Patellar reflexes sluggish, plantar responses normal.  
Upper limbs limp, particularly the left, the right  
could be raised with difficulty; unable to feed  
himself.  
Faucial angina and post nasal discharge noted.  
Attempts at lumbar puncture were unsuccessful.

Progress

Pyrexia lasted for 48 hours.  
Mental confusion and depression persisted for over  
two weeks.  
Sleep was very difficult.  
31:9:47. Paralysis of left deltoid evident, pre-  
ceded by tingling in the left arm.  
Pain in back and lower limbs persisted for the first  
two weeks, but no paralysis in these areas  
occurred.  
The paralysis coincided with involvement of the 5th  
and 6th cervical segments of the cord.  
29:10:47. Transferred to an E.M.S. Hospital for  
physiotherapy.

Treatment

During the first two weeks in hospital, continuous  
sedation was required; barbiturates were used to  
combat confusion and sleeplessness; and opiates  
were required to combat muscular pain.  
1:10:47. Abduction splint applied to left upper  
limb.

Contacts

On leave in Northamptonshire from about 15:9:47 to  
24:9:47.

FEMALE 28 Yrs. PARALYTIC.

Onset

26:9:47. Loss of appetite followed by vomiting.  
28:9:47. Headache, pain in the back of the neck.  
29:9:47. Nasal speech noticed.  
Reported and hospitalised.

On Admission T. 101.4. P. 100. R. 22.

Nasal intonation on speaking, left palatal paresis observed.

Rigidity of neck and spine slight.

Patellar reflexes sluggish, others apparently normal.

C. S. F. Slightly bloodstained.  
Under increased pressure.  
Lencocytes (all types) 60 per c. mm.

Progress

Pyrexia ceased after twenty-four hours.

1:10:47. Left facial paralysis, and sternomastoid.  
Difficulty in swallowing, sputum removed  
by spitting, no pharyngeal paralysis.

6:10:47. Palatal movement just present on left side.

8:10:47. Semi-solids swallowed.

10:10:47. Difficulty in getting rid of respiratory  
secretions.

17:10:47. Now able to swallow slowly.

Subsequent progress was uneventful, by the date of  
discharge, 1:11:47, the facial, sternomastoid, and  
palatal paresis had substantially improved, and  
she was able to go home.

The paralysis indicated brainstem involvement.

Treatment

The difficulty in swallowing and regurgitation  
necessitated artificial feeding: a tube passed  
into the oesophagus caused discomfort owing to  
constriction, and rectal feeding was adopted in  
preference.

The collection of mucus and the difficulty in deal-  
ing with respiratory secretions necessitated  
evacuation of these fluids by postural drainage.

Contacts

None to note.



MALE. 11 Yrs. NON-PARALYTIC.

Onset

28:9:47. Out of sorts, with pain in the back.  
29:9:47. Rigidity of neck and spine.  
Incontinence of urine.  
30:9:47. Reported and hospitalised at 12.30 a.m.

On Admission

Rigidity of neck and spine slight.  
No muscular pains.  
No faucial angina.  
Patellar reflexes - right present  
- left absent.  
No other reflex abnormality.

C.S.F. Not examined.

Progress

Incontinence of urine, resulting from retention,  
persisted till 1:10:47.  
Apyrexial after 1:10:47.  
Rigidity of neck and spine persisted till 3:10:47.  
29:10:47. Discharged after satisfactory convalescence.

Diagnosis

Rests on clinical evidence.

Treatment

On general lines.

Contacts

None to note.

MALE. 4 $\frac{1}{2}$  Yrs. PARALYTIC.

Onset

1:10:47. Listless and kept falling to one side.  
3:10:47. Pyrexia, rigidity of neck and pain in  
left arm.  
Reported and hospitalised.

On Admission T. 102.4. P. 96. R. 24.

Rigidity of neck and spine were present.  
Both upper limbs stiff, but moveable.  
Lower limbs unaffected.  
Twitching seen over the whole trunk, but most  
marked on the left side.  
Incontinent of urine.  
Plantar reflexes exaggerated, others apparently  
normal.

C. S. F. Under increased pressure.  
Slightly bloodstained with normal  
constituents.

Progress

Pyrexia continued for five days in the first week,  
and re-appeared for two days in the middle of the  
second week; this was not, in my opinion, the  
true biphasic effect.  
24:10:47. Right deltoid paralysed appeared suddenly  
and surprisingly after three weeks of satisfactory  
progress.  
This corresponded with involvement of the 5th and  
6th segments of the cervical cord.  
Discharged home on 5:11:47.

Treatment

This child was not severely ill and no special  
measures were required prior to the onset of the  
paralysis.  
An abduction splint was applied to the right upper  
limb.

Contacts

None to note.

FEMALE. 23 Yrs. NON-PARALYTIC.

Onset

29:9:47. Shivering, headache and pyrexia.  
4:10:47. After an interval of remission, symptoms  
recurred; patient was admitted to a  
general hospital and transferred to the  
fever hospital after examination.

On Admission T. 101.8. P. 110. R. 24.

Headache and photophobia severe.  
Tendency to fall back owing to weakness, but not  
paralytic.  
All voluntary movements present.  
No rigidity of neck or spine.  
Patellar reflexes absent, others normal.

C. S. F. Normal.

Progress

Pyrexia persisted for three days.  
5:10:47. Left ankle reflex absent, all other  
reflexes present and normal.  
29:10:47. Discharged after uneventful convalescence.

Diagnosis

This rests on clinical evidence of disease of the  
central nervous system, at a time of prevalence  
of poliomyelitis.

Treatment

General measures sufficed.

Contacts

This patient was of Polish Nationality and not very  
familiar with the English language. No history  
of contacts was elicited.

MALE. 2 Yrs. FATAL PARALYTIC CASE.

Onset

3:10:47. Appetite lost.  
4:10:47. Twitching of limbs and mouth.  
Incontinent of urine.  
Stated to have been unconscious for 4 hours  
Reported and hospitalised.

On Admission T. 99.4. P. 120. R. 26.

Right internal strabismus observed.  
Right leg flaccid.  
Rigidity of neck slight.  
A history suggestive of mental retardation was  
elicited.

C. S. F. Under increased pressure and bloodstained,  
no reliable information was obtained from  
its examination.

Progress

Pyrexia continued for three and a half weeks.  
5:10:47. Rigidity of neck and spine present.  
Patellar reflexes exaggerated.  
6:10:47. Right deltoid paralysis.  
10:10:47. Left internal strabismus.  
23:10:47. After two weeks of good progress, patient  
relapsed to-day with -  
Paralysis of muscles of back.  
Partial involvement of intercostal muscles.  
24:10:47. Both lower limbs now flaccid.  
Partial involvement of the abdominal  
muscles.  
Left facial paralysis.  
Sharp pyrexia.  
26:10:47. Both upper limbs now completely paralysed.  
7:11:47. Slight movement of hands.  
14:11:47. Partial movement of left forearm.  
Lower limbs completely flaccid.  
22:11:47. Transferred to the orthopaedic unit, as  
it was felt that more could be done for  
him there than in a fever hospital.  
4:12:47. After making promising progress, left-  
sided pneumonia set in and he died on  
14:12:47.

The distribution of the paralysis indicated involve-  
ment of practically the whole cord, and parts of  
the brainstem.

Treatment

This/



CASE 43 (Contd.)

Treatment

This proved to be a matter of difficulty owing to the daily change in the paralytic situation. In the orthopaedic unit he was treated in a complete plaster bed. Physiotherapy was difficult owing to persistent muscular tenderness.

Contacts

None to note.

FEMALE. 1 Yr. 2 Mths. PARALYTIC.

Onset

28:9:47. Teething, accompanied by coryza and drowsiness.

Vomited.

2:10:47. Limpness of the legs noted.

4:10:47. Reported and hospitalised.

On Admission T. 99. P. 132. R. 28.

Both lower limbs flaccid.

Patellar reflexes absent.

Rigidity of neck and spine present.

C.S.F. Under increased pressure, bloodstained, and its examination yielded no useful information.

Progress

Pyrexia remained for two and a half weeks.

6:10:47. Upper limbs noted to be limp.

Lower limbs still flaccid.

4:11:47. Transferred to the orthopaedic unit.

On arrival it was noted that the right lower limb was a complete flail, and that the muscles of the left lower limb were impaired.

The musculature of the abdominal wall was also impaired.

This corresponded with involvement of the lower thoracic, lumbar, and upper sacral portions of the cord.

Treatment

Abduction splints were applied to the lower limbs.

8:1:49. Discharged walking fairly well in callipers.

Contacts

One sister became a patient (Case 45).

Another sister, and a lodger, worked in Altyre

E.M.S. Hospital, where Case 35 had already occurred, on 19:9:47.

FEMALE. 8 Yrs. NON-PARALYTIC.

Onset

Fourteen days before admission, patient was discharged from a general hospital where she had undergone treatment for head injury. During those fourteen days, headache had been intermittent.

6:10:47. Headache accompanied by rigidity of neck and spine.  
Reported and hospitalised.

On Admission T. 101. P. 136. R. 26.

Headache severe.  
Rigidity of neck and spine marked.  
Tendon and cutaneous reflexes exaggerated.  
Head retraction, Kernig's sign elicited with manipulation of left lower limb but not right lower limb.  
Voluntary movements normal.

C.S.F. Lumbar puncture unsuccessful.

Progress

Pyrexia persisted for six days, slight biphasic effect.

7:10:47. Nauseated.

11:10:47. Apparently symptom free.

5:11:47. Discharged after normal convalescence.

Diagnosis

Rests on the clinical appearances coupled with the simultaneous occurrence of paralytic poliomyelitis in her sister (Case 44.)

Treatment

On general lines.

Contacts

Sister of Case 44.  
Remote contact of Case 35.

MALE. 13 Yrs. NON-PARALYTIC.

This patient was a pupil in a residential school and I am indebted for information to the school authorities and doctor, and the Medical Superintendent, City Hospital, Aberdeen.

Onset

5:10:47. Unwell, Pyrexial, isolated in school sanatorium.

7:10:47. Seen by consultant and removed to hospital.

C. S. F. Confirmed diagnosis of poliomyelitis, but details not given.

Progress

Satisfactory, no paralysis, discharged 25:10:47.

Diagnosis

Rests on clinical and pathological grounds.

Contacts

26:9:47. School term opened, patient was therefore in contact with other pupils for ten days, but no other case occurred, nor was there a suspected abortive case.

23:9:47. (approximately) Patient met in the street the brother of a female patient admitted to the City Hospital, Aberdeen, on 15:10:47. The brother was not ill at all. The Medical Officer of Health for Aberdeen observed that the contact was very remote. I agree but record the fact for what it is worth.



CASE 47.

MALE. 2 Yrs. NON-PARALYTIC.

Onset

7:10:48. Feverish.

8:10:48. Vomited, face twitched, very drowsy.  
Reported and Hospitalised.

On Admission T. 100.6. P. 156. R. 60.

Rigidity of neck and spine present.

Internal strabismus suspected, not seen on re-examination.

C.S.F. Contaminated with blood and not examined.

Progress

Pyrexial for 48 hours, with recurrence later,  
biphasic type.

23:10:47. Facial palsy suspected, but not seen on  
re-examination.

Other signs and symptoms were absent after two and  
a half weeks.

Diagnosis

Rests on clinical findings alone.

Treatment

On general lines.

Contacts

None to note.

FEMALE. 6 Mths. PARALYTIC.

### Onset

8:10:47. Vomited.

9:10:47. Drowsy and without appetite.

10:10:47. Reported and hospitalised.

On Admission T. 100.6. P. 120. R. 60.

Left facial paralysis present.

Left lower limb flaccid.

Patellar reflexes - left absent.

- right normal.

Plantar reflexes - left absent.

- right normal.

C. S. F.      Not examined.

Progress.

Pyrexia was present during the first week, and recurred during the third week.

The facial paralysis made feeding slow and difficult, but not impossible.

By the fourth week, the facial paralysis had greatly improved, but the flaccidity of the left lower limb was unch~~anged~~.

4:11:47. Transferred to the orthopaedic unit.

The location of paralysis indicated an extensive involvement of the lumbar and sacral cord, and slight brainstem involvement.

## Treatment

The lower limb was placed in an abduction splint.

The problem was to feed the child. Natural feeding fortunately proved adequate.

17:8:49. Still in orthopaedic unit, with supports.

## Contacts

None to note.

MALE. 11 Yrs. NON-PARALYTIC.

Onset

11:10:47. Pain in back of head, slight spinal rigidity.  
Reported and hospitalised.

On Admission T. 101.8. P. 120. R. 32.

Headache is aggravated by movement of the head.  
Rigidity of spine slight, of neck, not found.  
No reflex abnormality.

C.S.F. Under raised pressure, constituents normal.

Progress

Febrile for 3 days.

15:10:47. Spinal rigidity still present, and accompanied by lumbar pain for three days.

8:11:47. Discharged after satisfactory convalescence.

Diagnosis

Rests on clinical appearances and raised pressure of cerebrospinal fluid.

Treatment

On general lines.

Contacts

4:10:47 to 13:10:47, on holiday in Aberdeen.

MALE. 2 Yrs. FATAL PARALYTIC CASE.

Onset

12:10:47. Unwell, no specific complaints.  
13:10:47. No appetite.  
14:10:47. Vomited.  
15:10:47. Left facial palsy and strabismus.  
No other signs.  
Reported and hospitalised.

On Admission T. 101.4. P. 142. R. 36.

Facial palsy and internal strabismus.  
Slight impairment of movements of the soft palate.  
Rigidity of the neck and spine present.  
Rigidity of both upper limbs present.  
Tendon reflexes normal, cutaneous reflexes normal.  
Resents handling, and very irritable.

C.S.F. Not examined.

Progress

Incontinent of urine during the night.  
16:10:47. Early in the morning, patient became very  
short of breath and unable to swallow  
mucus.  
Respiratory muscles acting sluggishly.  
Vertical nystagmus noted intermittently.  
Temperature rose sharply.  
17:10:47. Died at 4.45 a.m.  
The sequence of events indicated involvement of the  
brainstem, with bulbar paralysis.

Contacts.

None to note.



FEMALE. 30 Yrs. NOT POLIOMYELITIS.

1:10:47. Stated she had weakness of legs and arms, pain in back, nausea.

14:10:47. Sensation of choking, unable to swallow. Reported and hospitalised.

On Admission T. 99.4. P. 108. R. 22.

No signs of paralysis, able to swallow.

This patient was acutely anxious and afraid of bulbar paralysis, she responded well to sedation.

Amended Diagnosis

Hysteria.

MALE.

11 Yrs.

NON-PARALYTIC.

Onset

16:10:47. Headache.  
18:10:47. Headache continued.  
Vomited.  
Rigidity of spine.  
Sore throat.  
19:10:47. Reported and hospitalised.

On Admission

T. 99.8

P. 70.

R. 20.

Frontal headache.  
Rigidity of neck slight.

C.S.F.

Pressure increased.  
Lymphocytes 50 per c.mm.  
Protein 50 mgm. per cent.

Progress

All symptoms and signs rapidly disappeared.  
No paresis appeared.  
Convalescence uneventful.  
Discharged 15:11:47.

FEMALE. 23 Yrs. DOUBTFUL.

Onset

19:10:47. Generalised malaise, vomiting.  
Temperature 100°F. Pulse 120.  
20:10:47. Reported and hospitalised.

On Admission T. 97.8. P. 88. R. 26.

Rigidity of spine present.  
Right patellar reflex absent.  
Abdominal pain slight.

Progress

Immediately after admission, the abdomen became very painful.  
Headache became severe.  
A diagnosis of acute appendicitis was made, and an inflamed appendix was removed.  
Pyrexia persisted for two days.  
Retention of urine necessitated catheterization for two days.

C. S. F. Owing to the appendicular condition, the cerebrospinal fluid was not examined.

After the first two days, neither abdominal nor nervous symptoms showed themselves.

Discussion

It is, of course, a pity that the cerebrospinal fluid was not examined, this might have put the matter beyond doubt.  
The question is, how much of the clinical picture was due to the appendicitis, and how much, if any, to poliomyelitis.  
There is only one symptom or sign in the case which could not in some degree have been brought about by the acute appendicitis.  
If the medical practitioner in charge of the case had not been experienced in assessing cases of poliomyelitis, the presence of spinal rigidity might well have been doubted.  
The interference with the right patellar reflex could result from muscular irritation by the inflamed appendix.  
The acute headache is the one sign not normally associated with an acute abdominal condition.

Contacts

None to note.

MALE. 9 Mths. NON-PARALYTIC.

Onset

20:10:47. Fretful, feverish, stiffness of neck and back.

21:10:47. Reported and hospitalised.

On Admission T. 99. P. 136. R. 30.

Rigidity of neck and spine.

Voluntary movements present and unrestricted.

Resistant to examination.

C.S.F. Not examined.

Progress

Pyrexia absent after 24 hours, recurred owing to intercurrent illness in the third week.

All signs of acute illness absent after 3 days.

22:11:47. Discharged, convalescence delayed by septic finger.

Diagnosis

Rests on clinical findings alone.

Treatment

On general lines.

Contacts

None to note.



CASE 55.

MALE. 2 Yrs. 6 Mths. NON-PARALYTIC.

Onset

24:10:47. Feverish, appetite poor.  
25:10:47. Stated to be unable to hold up head.  
Reported and hospitalised.

On Admission T. 101.6. P. 104. R. 26.

Rigidity of neck and spine present.  
Head retraction present, Kernig's sign not elicited.  
Patellar reflexes absent, others unaffected.

C. S. F.

Under increased pressure.  
Lymphocytes, 39 per c.mm.  
Protein 40 mgm. per cent.

Progress

26:10:47. Drowsy and resentful of handling.  
Pain in back.  
27:10:47. Tremor of right arm.  
Pyrexial for 5 days, slight biphasic  
effect.  
Rigid for four days.  
14:11:47. Walking inco-ordinate when first allowed  
up.  
Discharged after satisfactory convalescence.

Diagnosis

Rests on clinical and pathological findings.

Treatment

No special measures required.

Contacts

None to note.

MALE. 10 Mths. NOT POLIOMYELITIS.

Two months previously he had convulsions.

8:11:47. Admitted to a general hospital in convulsions, and had 16 since noon; transferred to fever hospital.

On Admission T. 99.4. P. 112. R. 28.

Admitted at 4.0 p.m., three fits between than and 6.0 p.m.

In convulsion, the child became cyanosed, rigid, with vertical nystagmus, dilated pupils. As the convulsion ended, the right leg was drawn up and the left twitched, thereafter remaining flaccid.

C. S. F. Pressure markedly raised.  
Cells, 3 per c.mm.  
Protein 40 mgm.

9:11:47. Convulsions ceased from 3.0 a.m.  
Movements of left leg normal.

22:11:47. Discharged after uneventful recovery.

Amended Diagnosis

Infantile convulsions associated with teething.

MALE. 11 Years. NON-PARALYTIC.

Onset

12:11:47. Headache and vomiting.

16:11:47. Lumbar puncture relieved headache.

21:11:47. Reported and hospitalised.

On Admission T. 98.8. P. 88. R. 24.

Headache.

Rigidity of neck and spine.

Patellar and abdominal reflexes exaggerated, plantar normal.

C. S. F. Specimen examined on 16:11:47 reported normal.

Progress

Afebrile after admission.

Headache, spinal rigidity and back pain were intermittently troublesome for 10 days.

20:12:47. Discharged after satisfactory convalescence.

Diagnosis

Based on clinical findings.

Treatment

General measures and hot applications to the painful back were sufficient.

Contacts

None to note.

CASE 58.

MALE. 3½ Mths. FATAL PARALYTIC CASE.

Onset

19:11:48. Pyrexia.

22:11:48. Reported with diagnosis of pneumonia and hospitalised.

On Admission T. 100.6. P. 110. R. 26.

The child was acutely ill.

Nasal catarrh and an enlarged gland in the neck, were found.

On auscultation, areas of bronchial breathing were detected in both sides of the chest.

In view of the diagnosis and clinical findings, penicillin therapy was commenced, the initial dose being 2000,000 units, followed by 50,000 units every three hours.

Progress

23:11:47. About 11 a.m. the child became acutely distressed with rapid and difficult breathing; there was difficulty in getting rid of sticky mucus from the throat.

Atropin, gr 1/250th, was given every four hours.

24:11:47. All feeds returned by the nose.

Respiratory difficulty still present, with in-drawing of the chest.

Examination of the throat revealed palatal paresis, and suggested pharyngeal paresis as well.

C. S. F. Under increased pressure.  
Normal constituents.

25:11:47. The muscles of respiration failed, hyperpyrexia set in and the child died at 3.15 a.m.

The findings indicated bulbar paralysis complicating acute bronchopneumonia.

Contacts.

None to note.



MALE. 6 Yrs. DOUBTFUL.

Onset

26:11:47. Coryza, loss of appetite, disturbed sleep.

30:11:47. Reported and hospitalised with alternative diagnoses - poliomyelitis or Pneumonia.

On Admission T. 102.2. P. 84. R. 28.

Headache severe.

Rigidity of neck and spine.

Kernig's sign present.

Patellar reflexes absent, others unchanged.

Cough present, both pulmonary bases dull, bronchial breathing reported.

C. S. F. Under normal pressure.  
Constituents normal.

Progress

The temperature fell over 3°F in 12 hours, and to subnormal in another 36 hours.

Under penicillin, the physical signs in the chest disappeared in 48 hours.

Similarly, the signs of irritation of the central nervous system abated in the first three days.

20:12:47. Discharged after uneventful convalescence.

Contacts

This patient and Case 60 were close contacts both at school and at home.

Discussion

The feature of this case which calls for comment is the marked degree of meningeal irritation in a relatively mild case of pneumonia.

If the respiratory findings be disregarded, the case was as clear-cut as many of those accepted as non-paralytic poliomyelitis.

The contact with another case is suggestive of poliomyelitis rather than pneumonia.

MALE. 5 Yrs. DOUBTFUL.

Onset

30:11:48. Rigors and vomiting.  
1:12:48. Headache and generalised aches and pains.  
Reported and hospitalised with alternative  
diagnosis - Pneumonia, meningitis,  
poliomyelitis.

On Admission T. 104.8. P. 136. R. 42.

Headache and photophobia.  
Rigidity of neck and spine.  
Generalised pain.  
Tremor of upper left trunk.  
Cough; dullness bronchial breathing and  
crepitations at the left pulmonary base.

C. S. F. Non-pathological.

Progress

Tepid sponging reduced the temperature by 3°F.  
immediately on admission.  
This temperature was subnormal after 36 hours,  
rose above normal twice more in the first week,  
and thereafter remained sub-normal.  
Signs in the chest and in the central nervous  
system disappeared in three days, the former in  
response to penicillin therapy.  
20:12:47. Discharged after uneventful convalescence.

Contacts

As for Case 59.

Discussion

In this case again, there was an undoubted  
pneumonia with concurrent meningeal irritation.  
If there had been no local prevalence of poliomyel-  
itis, a diagnosis of pneumonia and meningism  
would have been accepted without question.  
On account of local prevalence and the contact with  
another very similar and certainly simultaneous  
case, it cannot be denied that, on the standard  
of diagnosis of non-paralytic cases, a decision  
that this was a case of poliomyelitis complicated  
by pneumonia is as tenable as that of pneumonia  
and meningism.

MALE. 22 Yrs. NON-PARALYTIC.

Onset

29:11:47. Pains in the legs.  
3:12:47. Frontal headache severe.  
4:12:47. Reported and hospitalised.

On Admission T. 98. P. 70. R. 20.

Headache slight.  
Rigidity of spine but not of neck.  
Patellar reflexes exaggerated, others normal.

C.S.F. Not examined.

Progress

Apyrexial throughout, clearly worse prior to admission.  
29:12:47. Discharged after uneventful convalescence.

Diagnosis

This rests on minimal clinical evidence, and on the absence of evidence of any other condition.

Treatment

Routine measures.

Contacts.

None to note.

CASE 62.

FEMALE. 6 Mths. NOT POLIOMYELITIS.

5:12:47. 5.30 a.m. Respirations rapid, cyanosed.  
8.0 a.m. Vomited.  
Green Stools.  
9.0 a.m. Convulsion affecting face, jaw,  
with deviation of eyes to  
right.  
Patellar reflexes absent.  
4.0 p.m. Convulsion affecting left upper  
and left lower limbs, which  
were limp thereafter.  
No rigidity, Kernig's sign  
absent.  
Patellar and plantar reflexes  
absent on left.

On Admission T. 106. P. 146. R. 74.

5.50 p.m. Cyanosed, unconscious.  
Twitching of left side of body.  
Eyes deviated to the right.  
Left lower limb limp.

C.S.F. Blood-stained, not examined.

Frequent green stools.

Progress

Deteriorated rapidly, unable to swallow.  
Died 6:12:47. 2.30 p.m.

Amended Diagnosis

Gastro-enteritis.



FEMALE. 15 Yrs. DOUBTFUL.

Onset

7:12:47. Pain in side during the night, vomited.  
Stiffness and pain in back.  
Severe frontal headache.

On Admission T. 100. P. 98. R. 22.

Headache frontal, severe.  
Rigidity of neck and spine.  
Pain in right side of abdomen.

C. S. F. Non-pathological.

8:12:47. Several offensive bowel movements.  
Pyrexia and all other signs and symptoms  
gone.  
12:12:47. Recovery of Shigella Sonnei from faeces  
reported.  
Treated with succinyl-sulphathiazole.  
31:12:47. Faeces now clear of pathogenic organisms.  
Symptom-free. C  
Discharged.

Contacts

None to note.

Discussion

There can be no doubt that the patient sustained  
infection by a dysentery-producing organism  
The meningeal irritation was quite definite, and  
is not described as a usual accompaniment of  
dysenteric infections.  
In this case a diagnosis of non-paralytic poliomyel-  
itis would have been made had the dysenteric organ-  
isms not been recovered.

MALE. 4 Yrs. 6 Mths. DOUBTFUL.

Onset

- 13:12:47. Admitted to a general hospital for investigation of fit which occurred that day.  
17:12:47. Cerebrospinal fluid examined, non-pathological.  
21:12:47. Signs of meningeal irritation.  
Reported and transferred to Fever Hospital.

On Admission T. 97. P. 62. R. 20.

Very irritable, probable headache and photophobia.  
Rigidity of neck present but not marked.  
Kernig's sign not elicited.  
No reflex abnormalities.

Progress

- 22:12:47. Sharp rise of temperature in evening.  
No change in general condition.  
23:12:47. Temperature normal.  
No change in general condition.  
24:12:47. Pyrexial again this day, giving biphasic effect.  
Cough troublesome, otherwise improving.  
3:1:48. Discharged after satisfactory convalescence.

Subsequently this patient was in the Sick Children's Hospital, Aberdeen, on account of further fits. I am indebted to Professor Craig for particulars of his investigation, which may be summarized as follows:-

- 10:9:48. Admitted.  
25:9:48. Discharged.

Physical examination showed no abnormalities.

C. S. F. Under normal pressure.  
Non-pathological.  
X-ray of the skull showed no lesion.  
Wassermann reactions of blood and cerebrospinal fluid were both negative.  
No convulsions occurred in hospital and no cause was found for their previous occurrence.

Subsequently/

Subsequently Professor Craig commented that obscure cases of encephalitis occur, usually with an autumnal prevalence. Whether these cases were due to the virus of poliomyelitis or not, he was unable to say.

Discussion

There is little to add. The case could have been one of polioencephalitis, but this is neither substantiated nor disproved by the available evidence. The onset was at a time when cases of poliomyelitis had been recently prevalent, but the nearest case was a mile away, and occurred two months earlier. Other cases occurred at roughly the same time, but were much more distant. It was stated by the teacher of the school attended by the brother that he had been ill during the period of exclusion as a possible contact, and returned with a weak leg. This statement reached me about 18 months later. If correct it suggests that both this patient and his brother were true cases.

FEMALE.      5 Yrs. 6 Mths.      DOUBTFUL.

Onset

21:12:47.      Coryza.  
25:12:47.      Became hysterical, later in delirium  
for two days.  
28:12:47.      Troublesome cough.  
Rigidity of neck.  
Severe abdominal pain.  
Reported with alternative diagnosis of  
poliomyelitis or meningitis, and  
hospitalised.

On Admission      T. 102.      P. 120.      R. 26.

Rigidity of neck.  
Severe cough.

C. S. F.      Non-pathological.

No reflex abnormalities.

Progress

The temperature became normal in 12 hours and all  
the other symptoms vanished in that time.  
5:1:48.      Discharged having made a good recovery.

Discussion

There is little to be said.      This child had signs  
of an upper respiratory infection, accompanied by  
pyrexia and nervous phenomena out of all proportion  
to the severity of the respiratory condition.  
There is no direct evidence by which the cause of  
the nervous phenomena can be identified.      The  
occurrence took place at a time when there was or  
had recently been a prevalence of poliomyelitis,  
but at a place fairly remote from other cases.



MALE. 14 Yrs. DOUBTFUL.

Onset

7:1:48. Sent home from school on account of  
coryza and cough.  
Continued unwell all week.  
13:1:48. Severe headache and vomiting.  
Pain in back of head on movement.  
Reported with alternative diagnoses of  
poliomyelitis or meningitis.

On Admission T. 101.2. P. 86. R. 22.

Headache and nuchal rigidity.  
Regarded as case of influenza.

C.S.F. Not examined.

Progress

Temperature became normal in 24 hours, and rose four  
days later to give a suggestion of the biphasic  
effect.

15:1:48. Symptom-free.  
19:1:48. Discharged recovered.

Discussion

The recorded facts are quite compatible with the  
diagnosis of influenza, even to the biphasic  
pyrexia. On the other hand, the headache,  
vomiting and neck rigidity seem to have been  
excessive for so mild a case of influenza. There  
is nothing to prove or disprove either contention.

MALE. 7 Mths. DOUBTFUL.

Onset

24:12:47. Very fretful.  
Appetite impaired.  
No pyrexia, no tachycardia, no increase  
in respiratory rate.  
Conditions did not change for over a week.  
4:1:48. Reported with alternative diagnoses of  
Pink Disease, or Incipient Meningismus,  
and hospitalised.

On Admission T. 102.4. P. 160. R. 40.

The child was very fretful and cried continually.  
No neck rigidity, Kernig's sign not elicited.

C.S.F. Marked increased pressure.  
Non-pathological contents.  
Examination of faeces proved negative.

Progress

5:1:48. Neck rigid, Kernig's sign elicited.  
Green stools passed.

The temperature fell to normal in 48 hours and  
thereafter rose again for four days, returning  
to normal in the next three days.

By the time the pyrexia had left the patient, he  
was symptom-free.

28:1:48. Discharged.

Discussion

But for the occurrence of the green stools, I should  
almost certainly have included the case amongst  
those of non-paralytic poliomyelitis. Their  
occurrence suggests, as an alternative possibility,  
that there was meningeal irritation due to gastro-  
enteritis. The tentative diagnosis of Pink  
Disease can be disregarded.

FEMALE. 50 Yrs. Non-PARALYTIC.

Onset

- 10:2:48. Collapsed.  
Drowsiness and pain in shoulders and  
back of neck.  
14:2:48. Reported and hospitalised.

On Admission T. 97. P. 52. R. 12.

Rigidity of neck and spine.  
Retraction of head, Kernig's sign present.  
Headache severe, photophobia marked.  
Drowsy.

C. S. F. Under increased pressure.  
Constituents normal.

Progress

- 15:2:48. Pyrexial this and the following day, this  
could represent the second rise in  
temperature in a biphasic effect.  
18:2:48. Symptom free.  
24:2:48. Discharged after uneventful convalescence.

Diagnosis

Hysteria was suggested.  
In view of the clinical findings, which are consist-  
ent with poliomyelitis, I have included this case  
amongst those confirmed.

Treatment

Considerable sedation was required.

Contacts

None to note.

FEMALE. 11 Yrs. NON-PARALYTIC.

Onset

1:3:48. Sent home from school on account of  
headache and vomiting.  
Reported and hospitalised.

On Admission T. 101.6. P. 104. R. 26.

Headache present.  
Rigidity of neck and spine.  
Retraction of head, Kernig's sign present.

C. S. F. Lumbar puncture unsuccessful.

Progress

Pyrexia persisted for 48 hours.  
2:3:48. Vomited, other findings unchanged.

C. S. F. Under normal pressure.  
Normal constituents.

4:3:48. Symptom-free.  
9:3:48. Rigidity of spine made a transient re-  
appearance.  
27:3:48. Discharged after uneventful convalescence.

Diagnosis

Rests on clinical evidence alone.

Treatment

Penicillin and sulphathiazole were given by  
injection. Routine measures were otherwise  
sufficient.

Contacts

This patient lived with her grandmother in Nairn,  
her parents lived in a hutted camp on the out-  
skirts of Inverness.

During the previous weeks cases had occurred in  
the portion of Inverness-shire east of the Burgh  
and adjacent to Nairnshire.

No evidence of contact was obtained.



MALE. 8½ Yrs. NON-PARALYTIC.

Onset

27:2:48. Pain in legs, unable to stand.  
29:2:48. Headache and vomiting.  
Some bronchitis detected.  
2:3:48. Temperature 101°F.  
Patellar and ankle reflexes exaggerated.  
Abdominal reflexes - right side absent.  
- left side present.  
Rigidity of neck, Kernig's sign present.  
Reported and hospitalised.

On Admission T. 100.6. P. 98. R. 22.

Rigidity of neck not definite.  
Kernig's sign not elicited.  
Rigidity of spine marked.  
Patellar and ankle reflexes diminished.  
Abdominal reflexes absent.  
Spasm of muscles of thighs.  
Resented examination.

C. S. F. Normal pressure.  
Normal constituents.

Progress

Pyrexial for a week, maximum 48 hours after admission, definite biphasic effect.  
Improvement was steady from the time of admission.  
11:3:48. Spinal rigidity only remaining finding.  
26:3:48. Discharged.

Diagnosis

Based on clinical findings alone.

Treatment

Sulphathiazole 3 Grammes given before admission.  
Penicillin alone given after admission, total  
6,000,000 Units.

Contacts

This patient also comes from Nairnshire and is presumed to have been a victim from the east Inverness-shire outbreak.

CASE 71.

FEMALE. 8 Mths. NOT POLIOMYELITIS.

11:1:48. Cold and cough.

16:1:48. Reported as pneumonia and hospitalised.

On Admission T. 102. P. Uncountable. R. 46.

Signs and symptoms of acute pneumonia, dealt with accordingly.

18:1:48. Very drowsy.

19:1:48. Still drowsy.

Marked spinal rigidity.

C. S. F. Under slightly increased pressure.  
Constituents normal.

Subsequent progress satisfactory, discharged

31:1:48.

Amended Diagnosis.

Pneumonia and meningism.

FEMALE. 15 Yrs. PARALYTIC.

For the details of this case I am indebted to the Medical Officer of Health for the County Borough of Brighton.

Patient was on holiday in Lossiemouth from 15:8:47 to 15:9:47, with other members of her family. She returned to a boarding-school in Brighton on 17:9:47.

Onset

26:9:47. Vomited.  
27:9:47. Reported and hospitalised.

Progress

To quote the Medical Officer of Health for Brighton -

"She became rather ill, the alimentary tract and speech being affected: no muscular paralyses developed, however, and she gradually improved. She was discharged to a Convalescent Home on October 16th, quite recovered."

I have no hesitation in including this case in my series as the disease was clearly contracted in Lossiemouth.

She was in one of the larger hotels and had no apparent contact with any of the other cases which occurred about the same time.

The history given corresponds to a mild paralytic involvement of the brainstem.

MALE. 9 Yrs. NON-PARALYTIC.

Onset

18:3:48. Headache and vertigo.  
19:3:48. Headache severe.  
Faucial angina, epistaxis.  
Reported and hospitalised.

On Admission T. 100. P. 29. R. 24.

Faucial angina marked, breath offensive.  
Rigidity of neck.  
Retraction of head, kernig's sign elicited.

C.S.F. Not examined.

Progress

Afebrile after the first 12 hours.  
Spinal rigidity appeared after admission and was  
present for the remainder of the first week.  
14:4:48. Discharged after satisfactory convalescence.

Diagnosis

Rests on clinical findings.

Treatment

Routine measures sufficed.

Contacts

None to note.



CASE 74.

MALE. 32 Years. NOT POLIOMYELITIS.

1:7:48. Found unconscious.  
All reflexes normal.  
Kernig's sign doubtful.  
Reported and hospitalised.

On Admission T. 97. P. 80. R. 20.

Breath smelt of alcohol.  
No abnormalities elicited in nervous, respiratory,  
alimentary, or urinary systems.  
Subsequently it was learnt that he had attended a  
wedding and consumed a quantity of alcohol he  
could not tolerate.

2:7:48. Discharged.

Amended Diagnosis

Alcoholism.

FEMALE. 34 Yrs. NON-PARALYTIC.

Onset

26:7:48. Nausea.  
27:7:48. Vomited.  
Headache, feeling of weakness in neck  
and limbs.  
28:7:48. Reported and hospitalised.

On Admission T. 99.2. P. 98. R. 24.

Headache, vertigo, nausea.  
Rigidity of neck slight.  
Abdominal reflexes diminished, others apparently  
unaffected.

C. S. F. Under normal pressure.  
Lymphocytes, 95% of 440 cells per c.mm.  
Protein 90 mgm. per cent.  
Chlorides 700 mgm. per cent.

Progress

Pyrexia persisted for 4 days, slightly bi-phasic.

29:7:48. No change in nervous symptoms.

30:7:48. Developed swelling in the area of the  
left parotid gland.

Cerebrospinal Fluid re-examined -

Lymphocytes 300 per c.mm.  
Protein 40 mgm. per cent.  
Chlorides 670 mgm. per cent.

31:7:48. Rigidity has now gone.

Reflexes normal.  
Parotid swelling was not the only  
abnormality.

2:8:48. Steady clinical improvement from now on.

4:8:48. Cerebrospinal fluid re-examined -

Lymphocytes 500 per c.mm.  
Protein 120 mgm. per cent.  
Chlorides 700 mgm per cent, fine clot.

12:8:48. Cerebrospinal fluid re-examined -

Lymphocytes 115 per c.mm.  
Protein 50 mgm. per cent.  
Clear fluid.

17:8:48. Discharged after normal convalescence.

Diagnosis/

Diagnosis

For several days it was not clear whether this was a case of poliomyelitis or tuberculous meningitis. The clinical and pathological findings suited either diagnosis. The low chloride content of the specimen of C.S.F. secured on 30:7:48 tended to favour the latter diagnosis. The amelioration clinically, coupled with the slower improvement in the pathology of the C.S.F. served to rule out a tuberculous condition, and the diagnosis of poliomyelitis was therefore set up by exclusion. The parotid swelling which remained unilateral, deserves passing reference. Meningeal irritation occurs in Mumps, but in this condition there would not be the grossly abnormal C.S.F.

Treatment

Sedation, warm applications to the swollen parotid gland, and general measures sufficed.

Contacts

4:6:48. to 11:7:48. Staying in Dumfries.  
14:7:48. Attended a wedding in Aberdeen, and spent one night there; is stated to have been out of sorts from this date.

MALE. 54 Yrs. NOT POLIOMYELITIS.

Has not been well for some weeks, and has had occasional frontal headache, and lapses of memory.

26:7:48. Headache became severe and persistent.

28:7:48. Vomited.

Pain in back, neck, and right upper abdomen.

30:7:48. Reported and hospitalised.

On Admission T. 99. P. 84. R. 24.

Pain in back of neck.

Mentally confused.

Abdominal reflexes absent.

Tendon reflexes present in both upper and both lower limbs.

Tremor in the left upper limb.

Weakness in the left lower limb.

Kernig's sign not elicited.

C.S.F.

Clear.

Pressure normal.

Lymphocytes 60 per c.mm.

Protein 160 mgm per cent.

Chlorides 710 mgm. per cent.

Sterile.

### Progress

This was not satisfactory.

In view of the persistence of symptoms, a tentative diagnosis of tuberculous meningitis was made, and the case transferred to Aberdeen where streptomycin was available.

10:8:48. Patient died, and at post-mortem examination a large neoplasm infiltrating the corpus callosum was found. This was near enough to the lateral ventricle to induce the cell and chemical changes found in the cerebrospinal fluid.

### Amended Diagnosis

Cerebral neoplasm.



FEMALE. 55 Yrs. NON-PARALYTIC.

Onset

7:8:48. Stated to have swallowed fly.  
8:8:48. Temperature 102°F.  
Pain in back of neck.  
11:8:48. Reported and hospitalised.

On Admission T. 97. P. 96. R. 24.

Faucial angina present.  
Rigidity of neck present.  
Patellar and ankle reflexes diminished.  
Abdominal reflexes absent.  
Other reflexes normal.

C. S. F. Under increased pressure.  
Constituents normal.

Progress

Recovered rapidly.  
16:8:48. Discharged recovered.

Diagnosis

Rests on minimal clinical grounds, and the raised intrathecal pressure.

Treatment

Sedation and general measures.

Contacts

None to note.

MALE. 30 Yrs. NON-PARALYTIC.

Onset

24:8:48. "Influenzal symptoms."  
28:8:48. Headache acute.  
29:8:48. Reported and hospitalised.

On Admission T. 99.6. P. 86. R. 24.

Headache severe, accompanied by photophobia.  
Discomfort in lumbar region.  
Examination of nervous system negative.

C. S. F. Under normal pressure.  
Lymphocytes, 92% of 216 cells per c.mm.  
Protein 70 mgm. per cent.  
Chlorides, 725 mgm. per cent.  
No tubercle bacilli found then or by  
animal inoculation.

Progress

Pyrexia settled in 24 hours.  
A few moist sounds appeared at the base of the  
right lung.  
11:9:48. Discharged after satisfactory convalescence.

Diagnosis

Rests on minimal clinical findings, and pathological  
cerebrospinal fluid.

Treatment

On general lines.

Contacts

14:8:48 to 28:8:48, the date of onset of acute  
symptoms of patient carried out a motor tour to  
the Western and South Western Highlands, and the  
Firth of Clyde.

Two of this patient's employees were off work  
during this period on account of conditions not  
deemed to be poloimyelitis.

FEMALE. 39 Yrs.

Onset

10:8:48. (Approximately) Severe headache and giddiness.  
Rigidity of neck and spine were present for two weeks thereafter.  
31:8:48. Reported and hospitalised.

On Admission T. 98.6. P. 66. R. 24.

Not acutely ill.  
No rigidity of neck or spine.  
Tingling in left lower limb.  
Nausea and giddiness present.  
Reflexes present and normal.

C. S. F. Not under increased pressure.  
Constituents normal.  
Abdominal pain present.  
A few crepitations heard at the right pulmonary apex.

Progress

Straightforward convalescence in hospital, discharged 14:9:48.

15:10:48. Electrical reaction showed weakness of left Vastus Lateralis.  
22:10:48. Electrical reactions of left deltoid and rhomboids sluggish.  
These minimal pareses indicated slight involvement of the cervical and lumbar portions of the spinal cord. It is not clear when these transient pareses appeared. They responded to physiotherapy.

Contacts

From 5:7:48 to 12:7:48, this patient visited her sister and niece in Edinburgh. On 21:7:48, her niece was removed to the City Hospital, Edinburgh as a suspected case of poliomyelitis, on account of malaise, the legs being described as useless and heavy. The cerebrospinal fluid was normal. On 26:7:48, after purgation, Bacillus Dysenteriae Sonne was recovered from the faeces and the diagnosis amended accordingly. The dysentery rapidly cleared up with phthalyl-sulphathiazole. From the foregoing information, for which I am indebted to the Medical Superintendent, City Hospital, Edinburgh, it is clear that the niece did/

did not suffer from poliomyelitis, and that the patient's visit to Edinburgh is unlikely to have any relationship with her subsequent illness. This story has, however, led me to review the diagnosis of this case. The points in favour of a diagnosis of acute anterior poliomyelitis are the occurrence of headache, rigidity of neck and spine, altered sensations, giddiness and nausea, followed by transient and mild pareses of voluntary muscles. The points against this diagnosis are the prolonged nature of the illness, a minimum of six weeks having occurred between the onset and the pareses, and the normality of the cerebrospinal fluid. The first weighs much more with me than the second, as previous cases of paralysis with normal cerebrospinal fluid have been recorded in this study. At the same time I am bound to admit that, but for this time lag, the series of events would have been accepted as justification for the diagnosis. Several other cases in which the onset of paralysis has been delayed have been recorded in this study, and I have therefore, with some diffidence, decided to include this as a confirmed case of paralytic poliomyelitis with very late onset of paresis.



CASE 80.

FEMALE. 24 Years. NOT POLIOMYELITIS

For 10 days prior to admission this patient suffered from headache and nausea.

2:9:48. Reported and hospitalised.

On Admission T. 98. P. 80. R. 22.

Nausea still present.

No abnormalities elicited in the course of examination of the nervous system.

C. S. F. Under normal pressure.  
Constituents normal.

Pain in the lower abdomen.

Per vaginam, enlargement and tenderness of right ovary and tube.

8:9:48. Discharged.

Amended Diagnosis

Inflammation of ovary and fallopian tube.

FEMALE. 2 Yrs. DOUBTFUL.

Onset

Late in August, 1943, this patient's brothers took measles.

10:9:48. Measles rash in this case followed by a normal course for one week.

18:9:48. Meningeal irritation suspected.  
Reported and hospitalised.

On Admission T. 98.4. P. 106. R. 28.

The neck was thought to be rigid.  
Tendon reflexes were very difficult to elicit.  
Abdominal reflexes were exaggerated.

C. S. F. Non-pathological.

Progress

Pyrexia, with a maximum of 100°F. persisted for a week.

On one day the left lower limb seemed to be flaccid.

By the end of ten days the child was symptom-free.

7:10:45. Discharged.

Subsequently, electrical testing of muscles was attempted, unsuccessfully, as the child resisted equally effectively with all four limbs.

Discussion

There is evidence of meningism following measles.

Under the circumstances, the case is as likely to have been one of encephalitis of the post-exanthem type as poliomyelitis.

MALE. 12 Yrs. DOUBTFUL.

Onset

27:9:48. Vomiting and abdominal pain during the night.  
Headache, neck rigid.  
Reported and hospitalised.

On Admission T. 101. P. 124. R. 24.

Headache acute.  
Neck rigidity doubtful.  
Slight tenderness in the right ilias fossa, constipated.  
Patellar responses sluggish.  
Plantar reflexes flexor.  
Flexor responses in elbow and wrist sluggish.  
Abdominal reflexes present except in left lower quadrant.

C. S. F. Non-pathological.

Progress

Pyrexia absent after 36 hours.  
29:9:48. Abdominal reflexes quite brisk,  
Patellar and ankle reflexes sluggish.  
Elbow flexor absent.  
Diarrhoea commenced this day, and Salmonella Typhi Murium recovered from stools.  
Anomalies in the reflex responses continued till 4:10:48, giving a daily changing picture.  
16:10:48 Discharged no longer excreting pathogens and symptom-free.

Contacts

Son of Case 83, with whose case this one will be discussed.

FEMALE. 39 Yrs. DOUBTFUL.

Onset

26:9:48. Backache.  
27:9:48. Slight headache.  
Reported and hospitalised along with  
the preceding case.

On Admission .T. 100.8. P. 114. R. 26.

Headache, and pain in lumbar region.  
Slightly sick.  
Neck slightly rigid.  
Tendon reflexes normal.  
Abdominal reflexes absent.  
Nystagmus to the left.

C.S.F. Under increased pressure.  
Non-pathological.

Progress

Pyrexia was absent after 36 hours.  
1:10:48. ~~Salmonella Typhi~~ ~~Murum~~ recovered from stools.  
4:10:48 Neck rigidity and reflex abnormalities  
still present.  
18:10:48. Discharged.

Contacts

The simultaneous occurrence of an almost identical  
symptom-complex in her son has been noted.  
Her husband and daughter were unaffected.

Discussion

These two cases would almost certainly have been  
included in the group of non-paralytic poliomyel-  
itis cases but for the concurrent food-poisoning  
infection.  
Whether due to poliomyelitis or to toxæmia accom-  
panying the salmonella infection, there can be no  
doubt that both cases were due to the same set  
of aetiological factors.  
These cases are of the type in which there is a  
choice of diagnoses with nothing to prove which  
is correct.



FEMALE. 7 Years. DOUBTFUL.

Onset

28:9:48. Sent home from school on account of  
abdominal pain.  
Reported and hospitalised.

On Admission T. 99.8. P. 120. R. 24.

Child was very irritable and lay curled up in bed.  
All reflexes, tendon and cutaneous, very exaggerated.

Progress

Apyrexial after admission.  
All symptoms and signs rapidly abated after the  
bowel was washed out.

Contacts

None to note.

Discussion

This was probably a case of meningism resulting from  
toxaemia from a loaded bowel, and cured by the  
removal of the source of intoxication.

MALE. 6 Yrs. NON-PARALYTIC.

Onset

11:10:48. Headache and vomiting after a few days  
of coryza.

12:10:48. Reported and hospitalised.

On Admission T. 99.2. P. 70. R. 22.

Faucial angina mild.  
Rigidity of neck slight.  
Triceps reflexes exaggerated.  
Patellar reflexes very sluggish.  
Retention of urine.  
Intercurrent sarcoptic infestation.

Progress

14:10:48.

C. S. F.

Pressure normal.  
Lymphocytes 24 per c.mm.  
Protein 30 mgm. per cent.  
Sterile on culture.  
Fluid clear.

2:11:48. Discharged after uneventful convalescence.

Diagnosis

This rests on the clinical findings, the pathological findings, and the simultaneous occurrence of an identical illness in this patient's sister (Case 86).

Treatment

General measures sufficed after the skin had been treated with benzyl benzoate emulsion.

Contacts

Case 86 is this patient's sister.  
There is a possible, though remote, connection with Case 16, which will be discussed later.

CASE 86.

FEMALE. 8 Yrs. NON-PARALYTIC.

Onset

11:10:48. Headache and vomiting.

12:10:48. Reported and hospitalised.

On Admission T. 97. P. 82. R. 20.

Virtually symptom-free except for sarcoptic infestation.

Progress

14:10:48.

C. S. F.

Clear fluid.

Pressure normal.

Lymphocytes 26 per c.mm.,

Protein 30 mgm. per cent.

Asymptomatic while in hospital.

2:11:48. Discharged.

Diagnosis

This rests on the pathological C.S.F., a history of headache and vomiting, and the simultaneous attack in her brother. None of the cases accepted on clinical grounds alone was as free from symptoms and signs of poliomyelitis as this patient.

Treatment

Treatment of sarcoptic condition sufficed.

Contacts

The same as for Case 85.

MALE. 25 Yrs. NON-PARALYTIC.

Onset

Headache for the previous 3 weeks.

22:10:48. Vomited.

23:10:48. Reported and hospitalised.

On Admission T. 97.8. P. 54. R. 18.

Rigidity of neck present.

Headache.

Lumbar puncture unsuccessful.

Patellar reflexes sluggish.

Abdominal reflexes, left upper quadrant absent.

Progress

Apyrexial throughout.

Signs referable to the nervous system rapidly subsided.

26:10:48. C.S.F. now secured and examined -

Pressure normal.

Clear.

Lymphocytes 140 per c.mm.

Protein 60 mgm. per cent.

Tubercle bacilli not seen.

3:11:48. Discharged after normal convalescence.

Treatment

On general lines.

Contacts

None to note.

FEMALE 1 Yrs. 10 Mths.

NOT POLIOMYELITIS.

22:10:48. Rash of chickenpox noted.

26:10:48. Neck rigidity and irritability present.  
Reported and hospitalised.

On Admission T. 95. P. 120. R. 38.

Pale and listless.

Resented examination.

Lesions of chickenpox on face, head and trunk.

Tendon and cutaneous reflexes not altered.

Held herself rigid.

Abdomen distended.

C. S. F. Under normal pressure.  
Constituents normal.

Progress

27:11:48. Discharge delayed on account of sepsis  
of skin lesions.

Amended Diagnosis

Chickenpox complicated by sepsis.



MALE. 25 Yrs. PARALYTIC.

For particulars of this case I am indebted to the Medical Superintendent, Culduthel Hospital, Inverness. His home address is in Buckie, Banffshire, but he was working near Aviemore, Inverness-shire, and is only included in the series because of his short stay in hospital in Grantown-on-Spey, Morayshire, where the diagnosis was established.

Onset

12:11:48. Pain in the chest, attributed to fibrositis.  
14:11:48. Severe headache and pain in the back.  
Hospitalised in Grantown-on-Spey.  
16:11:48. Pain in the left leg.  
17:11:48. Weakness in left leg.  
Transferred to Culduthel Hospital,  
Inverness.

On Admission

Retention of urine for 24 hours.  
Upper limbs and thorax unaffected.  
Weakness of lower recti abdominis, and erector spinal.  
Left lower limb flaccid with the following exceptions --

Knee flexors - faint contraction.  
Knee extensors - faint contraction.  
Foot, intrinsic muscles - faint contraction.

Right lower limb --

Hip muscles almost completely paralysed.  
knee flexors contracted against no resistance.  
Knee extensors contracted against gravity.  
Leg muscles strong except those giving the movement of inversion.

C. S. F. Lymphocytes 92% of 194 cells per c.mm.  
Protein 150 mgm. per cent.

Progress

Retention of urine for four days.  
Pain in the legs was intermittently present.  
8:12:48. Transferred to an orthopaedic unit for physiotherapy but with no improvement.  
The paralysis corresponds with lumbo-sacral involvement of the cord.

Treatment/

Treatment

Catheterisation was necessary for four days after admission.

The legs were splinted in the optimum position.

Contacts

Another case occurred in the Aviemore area with onset on 20:12:48. This patient, a male of 28 years, sustained paralytic poliomyelitis, the cerebrospinal fluid containing no cells, and protein 60 mgms. per cent. There was no known contact between the two cases, and the interval between the dates of onset suggests that any contact was by way of carriers or missed cases. This second case had no connection with Moray & Nairn and is not included in the series.

FEMALE. 15 Yrs. NON-PARALYTIC.

Onset

28:11:48. Abdominal pain and headache.

29:11:48. Vertigo.  
Admitted from a residential institution.

On Admission T. 97.6. P. 84. R. 22.

Headache and vertigo.  
Rigidity of neck and spine not present.  
Abdominal reflexes absent.  
Triceps reflexes present.  
Patellar reflexes - right, sluggish.  
left, absent.

C. S. F. Clear.  
Under increased pressure.  
Constituents normal.

Progress

Apyrexial throughout.  
30:11:48. No neck rigidity or spinal rigidity.  
Triceps reflexes both normal.  
Abdominal reflexes - upper quadrants sluggish.  
- lower quadrants absent.  
Patellar reflexes both absent.  
Plantar response flexor.

C. S. F. Under increased pressure.  
Blood-stained and not examined.

2:12:48. Headache.  
Patellar reflexes absent.  
Abdominal reflexes present.  
3:12:48. Headache and photophobia severe.  
5:12:48. Headache and photophobia decreasing.  
Reflexes normal.  
14:12:48. Discharged.

Diagnosis

Rests on the symptoms, the persistent alterations in the tendon and cutaneous reflexes, and in the raised pressure of the cerebrospinal fluid.

Treatment

Sedation and general measures.

Contacts

No other case, or possible abortive case, is known to have been connected with this patient.

Two further cases of non-paralytic poliomyelitis fall to be considered here. In both the initial diagnosis was suspected cerebrospinal meningitis.

MALE. 12 Yrs.

16:12:48. Illness commenced with headache.

21:12:48. Vomited.

Reported and hospitalised.

On Admission T. 99.6. P. 66. R. 18.

Frontal headache and photophobia.

Rigidity of neck.

C. S. F.

Clear

Under normal pressure.

Lymphocytes 72 per c.mm.

Protein 65 mgm. per cent.

Sterile.

Progress

Symptoms did not decrease, but became more marked with drowsiness and head retraction.

As the cerebrospinal fluid result did not exclude the diagnosis of tuberculous meningitis, it was decided to transfer him to Aberdeen where streptomycin was available, and this was done on 24:12:48. The diagnosis of tuberculous meningitis was confirmed in Aberdeen after the lapse of three or four days.

Amended Diagnosis

Tuberculous meningitis.

MALE. 42 Yrs. NON-PARALYTIC.

This is my own case, which is the only excuse for giving it in such detail.

Onset

3:6:48. Pyrexia with rigor.  
Lassitude.  
Slight headache.

Progress

4:6:48 Pyrexia declining.  
Lassitude present.

6:6:48 Walked half-a-mile for Sunday papers and to get car from garage.  
In afternoon, intense vertigo and nausea set in, with moderate headache.

7:6:48 Attended Committee Meeting, which brought on waves of nausea and vertigo.  
Lassitude and difficulty in starting movements.  
Sought medical advice, found to have -  
  
Patellar and ankle reflexes exaggerated and equal.  
Abdominal reflexes almost absent.  
Nystagmus to the right.  
Fundi normal.

An obstinate determination to continue my normal activities was present up to this date.

8:6:48 Rigidity of spine transient.  
Pain in lumbar region.  
Hyperaesthesia in right thigh.  
Dysarthria, occasional stumbling over words.

C. S. F. Not examined.

After Effects

The vertigo passed off in about three weeks, but occasionally returns even now on turning sharply to right.  
On going about, considerable unsteadiness was experienced.  
Mental depression persisted for three to four months.  
Migraine-type headaches were much more severe during/



during the subsequent nine months.

Diagnosis

Based on the neurological findings which were similar to those in other cases where the diagnosis was accepted.

MALE. 60 Yrs. NON-PARALYTIC.

Date May, 1948.

Presenting Symptoms Vertigo.  
In other respects similar  
to Case .

C.S.F. Not examined.

Not admitted to hospital.

Recovery after slow convalescence.

CASE 94.

FEMALE. 65 Yrs. NON-PARALYTIC.

Date May, 1948.

Presenting Symptom Vertigo.

This was the most severe case in respect of vertigo, which persisted even in the recumbent position.

Spinal rigidity and anomalous reflexes were present.

C. S. F. Not examined.

Not admitted to hospital.  
Recovery satisfactory.

MALE. 42 Yrs. NON-PARALYTIC.

Date October, 1948.

Presenting Symptom Vertigo.

This made standing and walking impossible for about three days, but did not persist while patient lay down.

Spinal rigidity and anomalous reflexes were present.

C.S.F. Not examined.

Not admitted to hospital.

Recovery satisfactory.

MALE. 28 Yrs. NON-PARALYTIC.

Date November, 1948.

Presenting Symptom Vertigo.

This was marked when the patient stood up, but did not prevent walking.

Patient slept almost continuously for 36 hours after vertigo first appeared and woke up much better.

C.S.F. Not examined.

Not admitted to hospital  
Rapid and satisfactory recovery.



CASE 97.

MALE. 60 Yrs. NON-PARALYTIC.

Date June, 1948.

Presenting Symptom Vertigo.

This was of average severity, making standing and walking difficult.

Reflexes were anomalous.

Slight rigidity of spine.

C.S.F. Not examined.

Not admitted to hospital.

Satisfactory recovery.

(Fraser McNeill).

MALE. 4 Yrs. NOT POLIOMYELITIS.

Onset

6:4:49. Neck stiff and painful.  
Eyelids swollen.  
No vomiting.

On Admission T. 98. P. 100. R. 20.

Rigidity of neck slight.  
Faucial angina marked.  
Enlarged cervical glands.

C.S.F. Pressure normal.  
Constituents normal.

Progress

10:4:49. Diagnosis amended to tonsillitis.

CASE 99.

MALE. 1 Yr. 4 Mths. DOUBTFUL.

Onset

13:4:49 Headache presumed, pyrexial.  
Vomited.  
Very fretful.

C.S.F. Not pathological.

Case treated at home, making a good recovery.

Diagnosis

There was undoubtedly a fairly severe pyrexial condition with excessive irritability. Nothing was elicited which justified a diagnosis of poliomyelitis. Equally, the findings were not wholly incompatible with this diagnosis.

(Jean Mitchell).

CASE 100.

FEMALE. 15 Yrs. PARALYTIC.

Onset

25:5:49. Headache and pyrexia.

30:5:49. Headache severe.

Numbness in toes and fingers.

Reported and hospitalised.

On Admission

T. 98.6. P. 90. R. 20.

Rigidity of neck slight, of spine absent.

Reflexes normal.

Pain in back and right arm.

Ocular fundi normal.

C.S.F.

Cells 0

Protein 90 mgm per cent.

Pressure raised.

Progress

1:6:49. Blurred vision, and transient nystagmus.  
paresis right side of face.

3:6:49. Paresis of small muscles of neck.

6:6:49. Paresis of intercostal muscles.

8:6:49. Paresis of most of right lower limb.

18:6:49. Paresis of left foot - foot dropped.

19:7:49. All pareses much improved.

Discharged to orthopaedic unit.

Paralysis indicated involvement of nearly the whole  
cord and brainstem.

Treatment

General measures sufficed.

Contacts

None to note.

(John Logan)

CASE 101.

MALE. 5 Yrs. NON-PARALYTIC.

Onset

17:6:49. Pyrexial.

18:6:49. Headache and vomiting.

22:6:49. Reported and hospitalised.

On Admission

T. 98.4. P. 80. R. 20.

Headache.

Rigid neck.

No other abnormalities.

C. S. F.

23:6:49. Lymphocytes 48 per c.mm.

Protein 55 mgm. per cent.

Sterile, no T.B. found on  
animal inoculation.

25:6:49. Lymphocytes 89% of 320 per c.mm.

Protein 70 mgm. per cent.

Sterile.

Diagnosis

Rests on clinical signs and pathological C.S.F.

Progress

No paralysis developed.

13:7:49. Discharged home.

Contacts

None to note.



(James R. Young).

CASE 102.

MALE. 1 Yr. 9 Mths. PARALYTIC.

Onset

10:7:49. Pyrexia, rigor, and jerky.

12:7:49. Left upper limb paralysed.

13:7:49. Reported and hospitalised.

On Admission

T. 99. P. 144. R. 36.

Flaccid paralysis of left shoulder and arm.  
Fingers slightly moveable.

C. S. F.

Lymphocytes 4 per c.mm.

Protein 40 mgm. per cent.

Examination not repeated.

Progress

23:7:49. Urticarial rash.

19:8:49. Transferred to orthopaedic unit.

Treatment

Abduction splint.

Contacts

3:7:49. Went for picnic, badly sunburned.  
Very tired after outing.

(Anne Arbuthnot)

CASE 103.

FEMALE. 15 Yrs. NOT POLIOMYELITIS

Onset

20:7:49. Suddenly developed stiff neck and right arm. when preparing to go to work.  
Reported and hospitalised.

On Admission. T. 97.8. P. 72. R. 24.

Neck stiff on right side, remained so for 24 hours.

Progress

It soon became evident that this was a case of fibrositis. Further investigation at home indicated unhappiness at work.

29:7:49. Discharged well.

(John Geddes)

CASE 104.

MALE. 24 Yrs. PARALYTIC.

Onset

30:7:49. Lassitude.  
31:7:49. Headache.  
1:8:49. Vomited.  
Pain in back.  
Paresis of right arm.  
2:8:49. Vertigo.  
Reported and hospitalised.

On Admission

T. 97.4. P. 104. R. 24.

Pain in back.  
Pupils unequal.  
Paresis of right hand, forearm and arm, limited  
movement of right shoulder.  
Paresis of left hand and forearm, slight.

C.S.F.

Pressure raised.  
Lymphocytes 90% of 86 cells per c.mm.  
Protein 90 mgm. per cent.

Progress

3:8:49. Left otitis media developed.  
Mentally confused.  
12:8:49. Mental condition normal.  
Otitis media cured.  
No further paresis.  
31:8:49. Still in isolation hospital.

Treatment

Sedation.  
Splinting not required.

(Sinclair Thomson)

CASE 105.

MALE      6 Yrs.      NOT POLIOMYELITIS.

Onset

6:8:49.    Sore throat and pyrexia.

11:8:49.    Slight scarlatiniform rash.  
Legs stiff.

16:8:49.    Arms and neck stiff.  
Reported and hospitalised.

On Admission

T. 101.8.    P. 112.    R. 34.

Faint scarlatiniform rash on chest.

Tongue - white strawberry.

Tonsils inflamed.

Pains in arms and legs.

Diagnosis Amended to Streptococcal Tonsillitis.

(Lindsay Allan)

CASE 106.

MALE. 4 Yrs. NOT POLIOMYELITIS.

Onset

7:8:49. Child very tired.  
Rigidity of neck slight.  
No head retraction.  
Kernig's sign stated to be positive.  
Headache.

9:8:49. Reported and hospitalised.

C.S.F. 7:8:49. Noth pathological.

On Admission T. 99.6. P. 120. R. 24.

Very flushed.  
Headache.  
Pupils unequal.  
Rigidity of neck slight.

Treatment

Penicillin 200,000 U. T.I.D.

Progress

Rapid recovery.  
Amended diagnosis, influenza.  
24:8:49. Discharged.

Contacts

None to note.



(Florence Dunbar)

CASE 107.

FEMALE.      7 Yrs.      NOT POLIOMYELITIS.

Onset

Off colour for six weeks.

Pain in back for 1 week.

Vomiting 1 week.

16:8:49. Admitted to Cottage Hospital.

17:8:49. Transferred to I.D. Hospital.

C.S.F.      16:8:49. Lymphocytes 20 per c.mm.  
Protein 35 mgm. per cent.

On Admission      T. 100.      P. 100.      R. 36.

Headache in frontal area.

Pain over site of L.P.

Pain in back on forward flexion of head.

Right patellar reflex reduced.

Progress

18:8:49. Tubercle bacilli found on repeated  
examination of C.S.F.

Transferred for Streptomycin.

Amended Diagnosis

Tuberculous meningitis.

(Donald Webster)

CASE 108.

MALE. 10 Yrs. NOT POLIOMYELITIS.

Onset

18:8:49. Pain in side.

Nausea.

Severe headache.

20:8:49. Reported and hospitalised.

On Admission

T. 102. P. 128. R. 28.

Child very flushed.

Pain in abdomen.

Pain in back of neck.

Neck perhaps rigid.

Throat inflamed.

Progress

This case rapidly showed itself to be one of  
tonsillitis and equally rapidly yielded to  
appropriate treatment.

27:8:49. Discharged.

(Marlene Weir)

CASE 109.

FEMALE. 3½ Yrs. PARALYTIC.

Onset

Not well for previous fourteen days.

17:8:49. Loss of appetite.

20:8:49. Reported and hospitalised.

On Admission

T. 98.6. P. 112. R. 22.

Left Patellar reflex absent.

Left leg partially paralysed.

Pain in left foot.

Rigidity of neck.

C. S. F. Normal findings.

Progress

22:8:49. Very irritable.

26:8:49. Pain on movement of left ankle.

29:8:49. General condition normal.

31:8:49. In hospital.

Treatment

Sedation.

Cage to prevent drop-foot.

(Robt. Smith)

CASE 110.

MALE. 2 Yrs. DOUBTFUL.

Onset

21:8:49. Gastro-intestinal upset for five days.  
Vomiting.  
Constipation.  
Pyrexia.  
Weakness thought to be present in left  
quadriiceps femoris.  
Reported and hospitalised.

On Admission T. 99.4. P. 120. R. 28.

No neuro-muscular abnormality.

C.S.F. Normal pressure.  
Normal constituents.

Progress 31:8:49. Still under observation.  
No abnormality since admission.

Diagnosis

History consistent with a diagnosis of poliomyelitis  
with transient paresis, but without any confir-  
mation.

CHART No: I.



# WORLD DISTRIBUTION OF POLIOMYELITIS

CHART No: I.

1939 TO 1945.

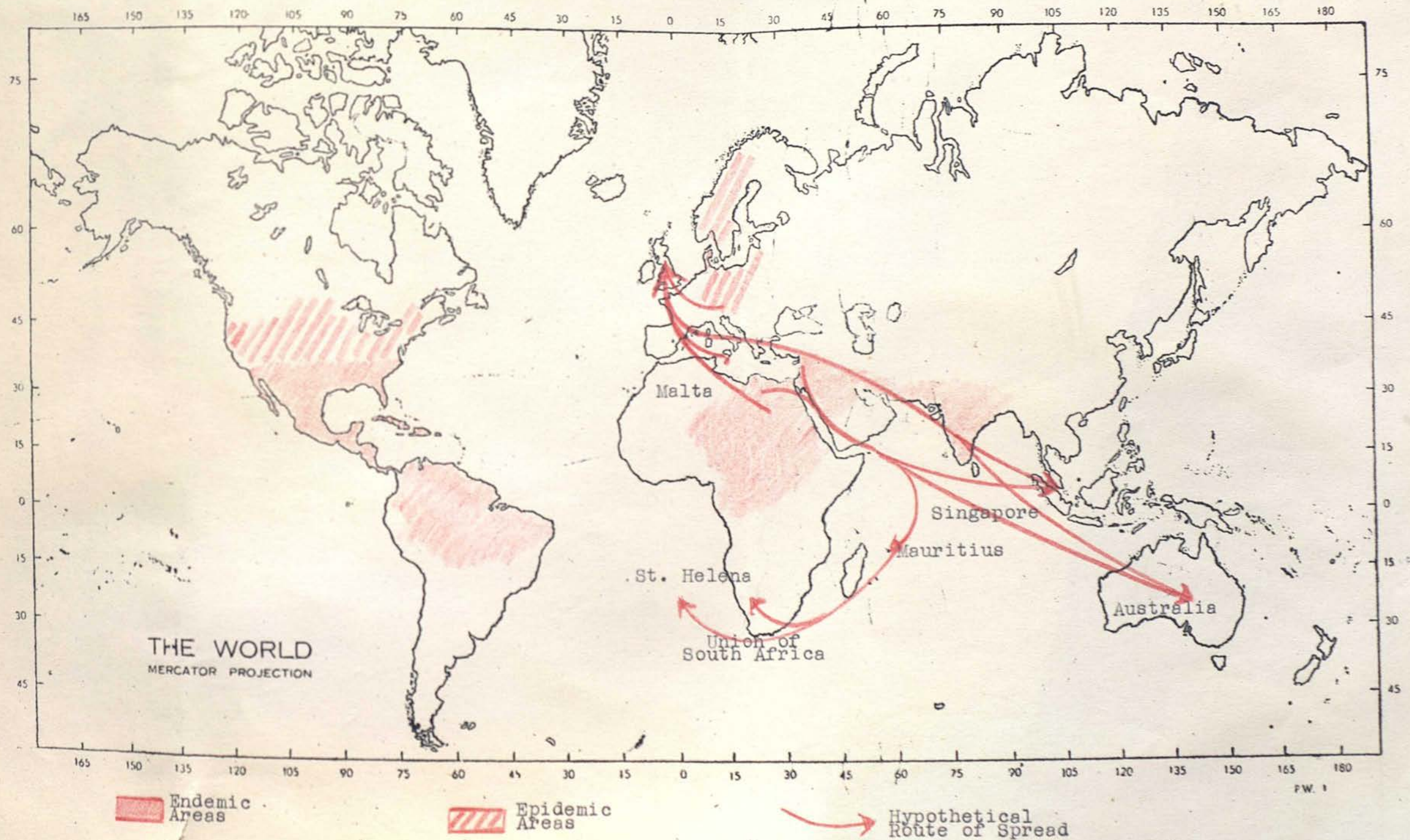


CHART No: II.



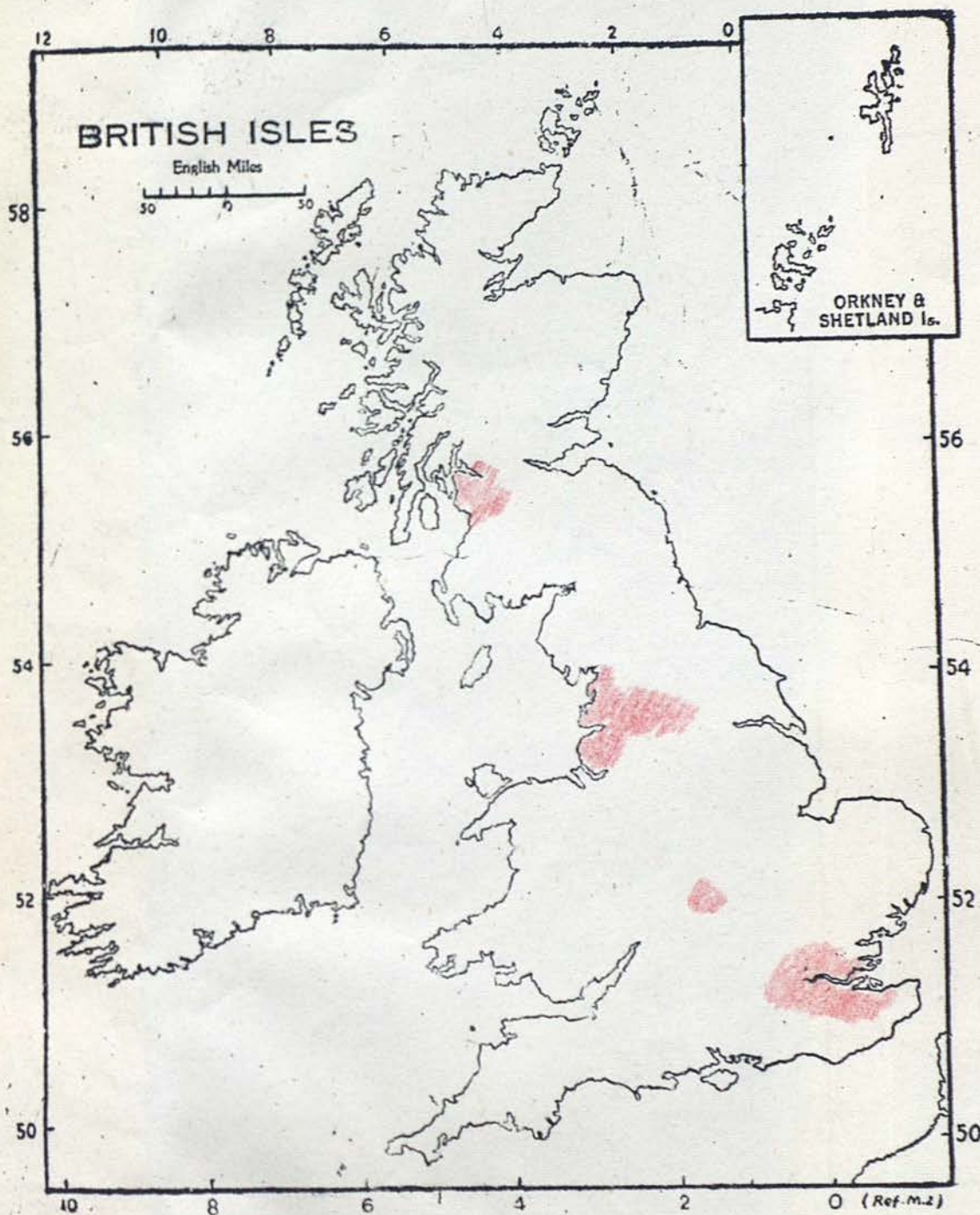
POLIOMYELITIS IN GREAT BRITAINAREAS OF PREVALENCE IN THE  
EARLY WEEKS OF 1947.

CHART No: III.



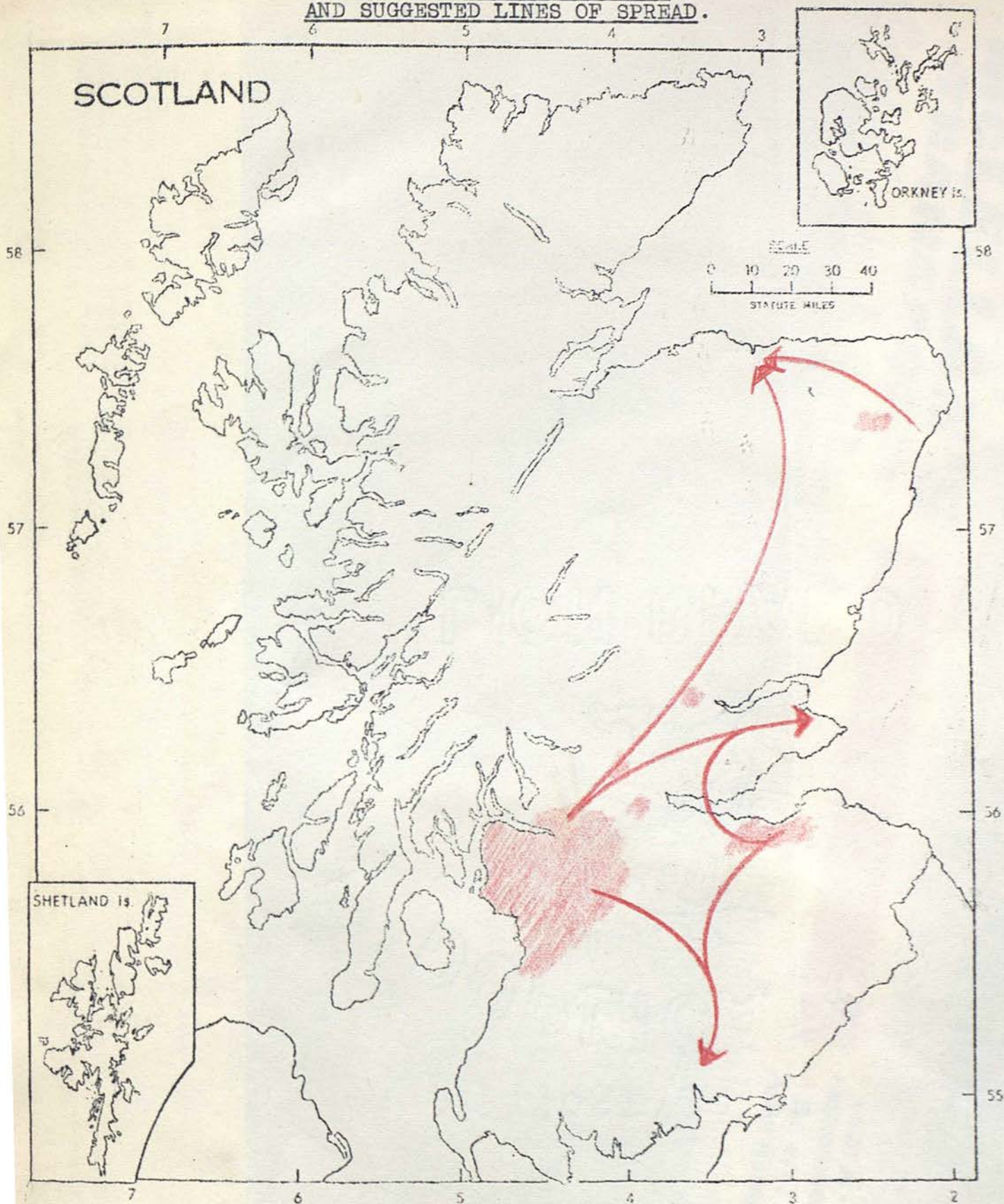
AREAS OF EARLY PREVALENCE  
AND SUGGESTED LINES OF SPREAD.



CHART No: IV.



# MORAY AND NAIRN

PHYSICAL FEATURES  
AREAS OF PREVALENCE OF POLIOMYELITIS

CHART No: IV.

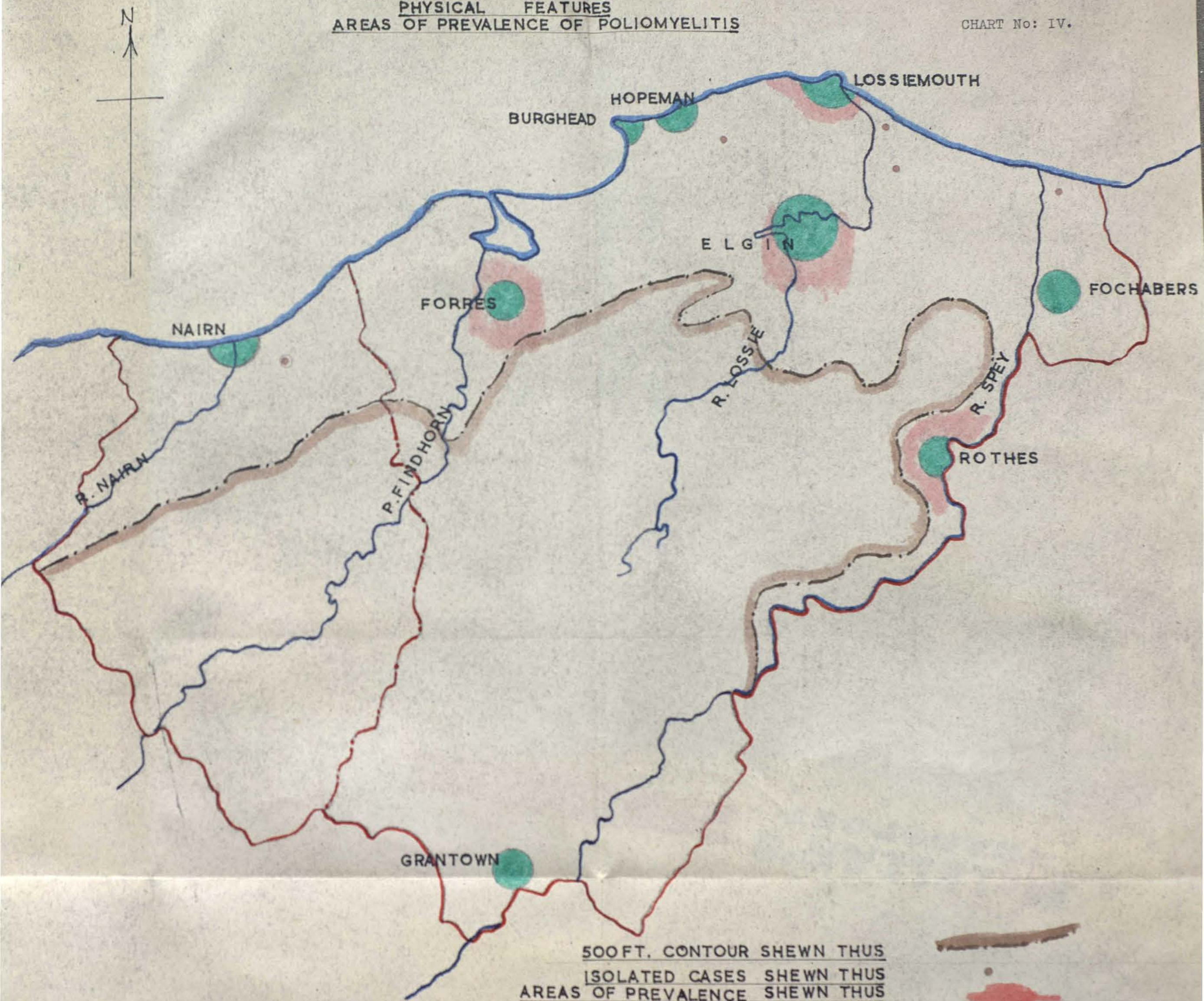




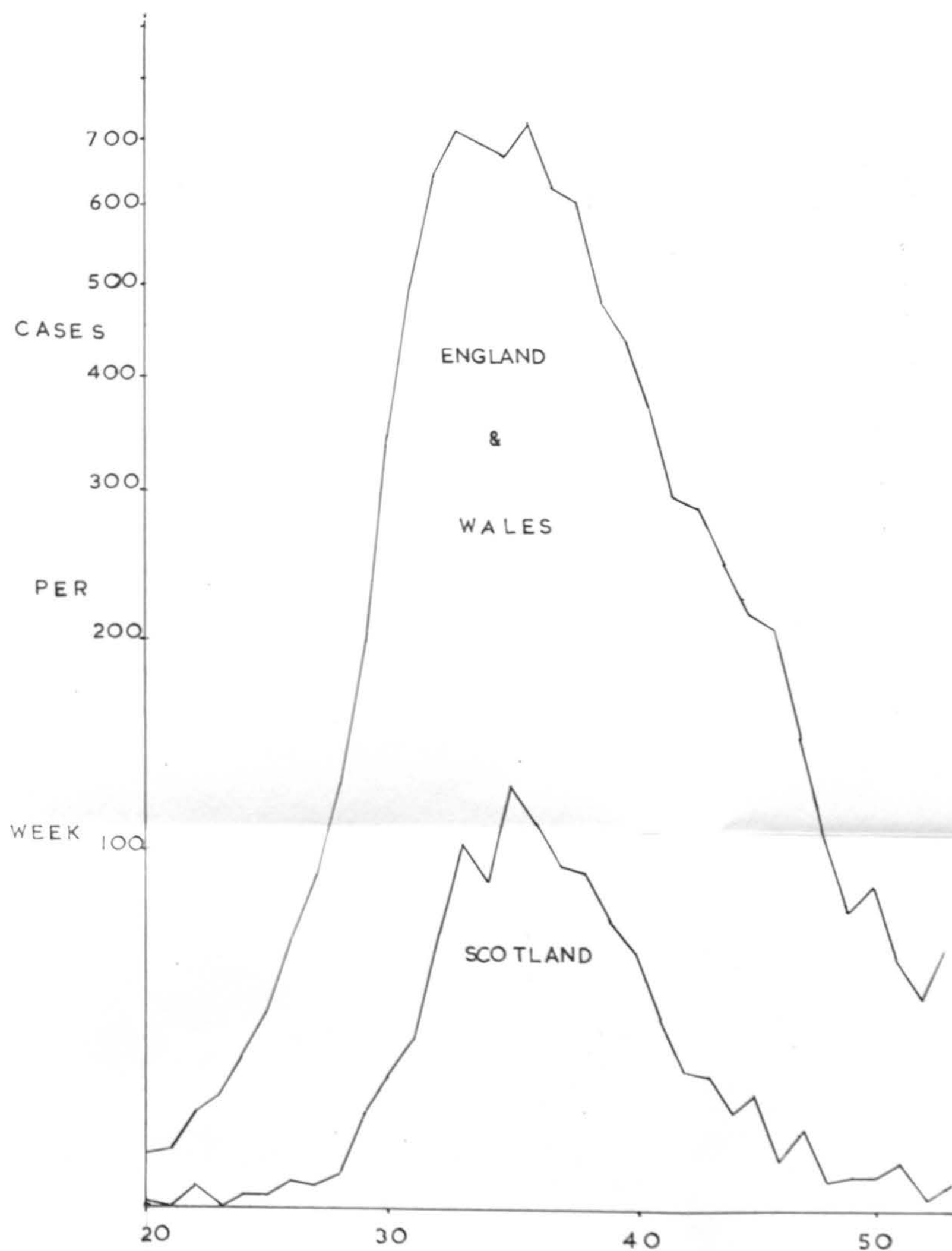
CHART No: V.

# NOTIFICATIONS OF POLIOMYELITIS 1947.

ENGLAND AND WALES

COMPARED WITH

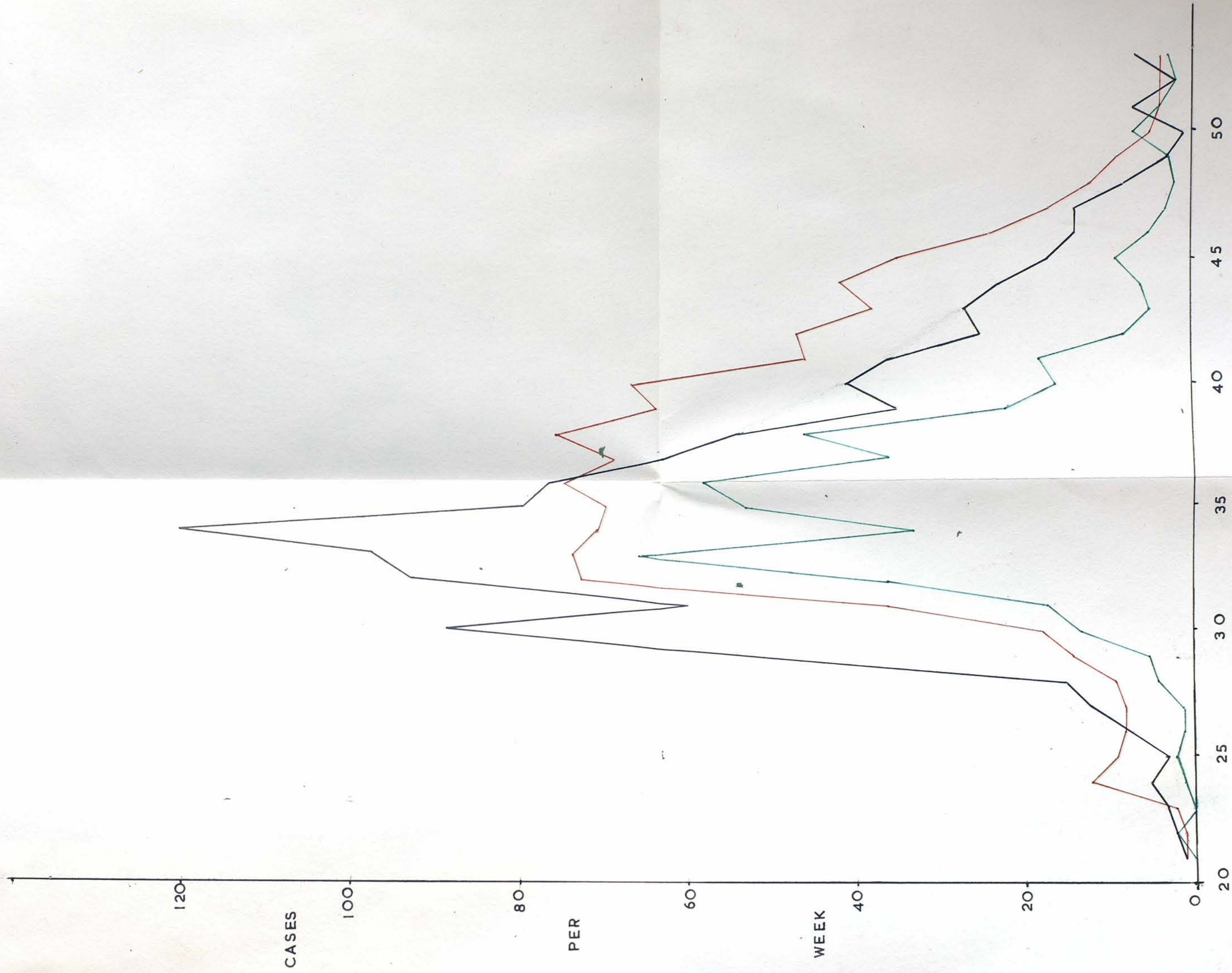
SCOTLAND.



WEEK OF YEAR 1947.

# NOTIFICATIONS OF POLIOMYELITIS 1947.

LONDON LANCASHIRE AND GLASGOW.



WEEK OF YEAR 1947.



CHART No: VI.

CHART No: VII.

POLIOMYELITIS IN FIVE SCOTTISH AREAS 1947.

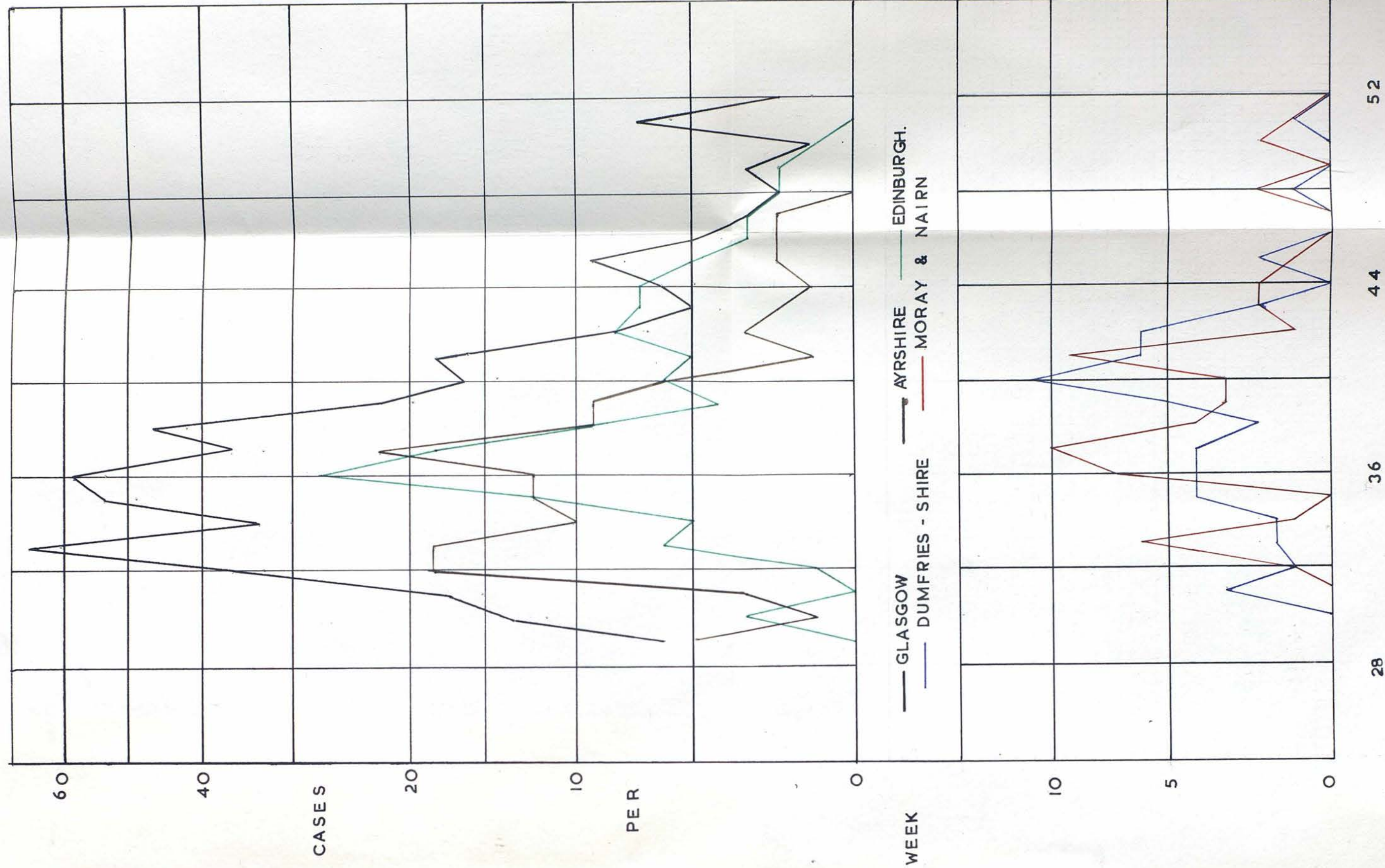


CHART No: IX.

# TRAVELLING IN RELATION TO ONSET OF POLIOMYELITIS

